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A Practical Journal on Psychiatry and Neurology

(CONTENTS ON PAGE 180)

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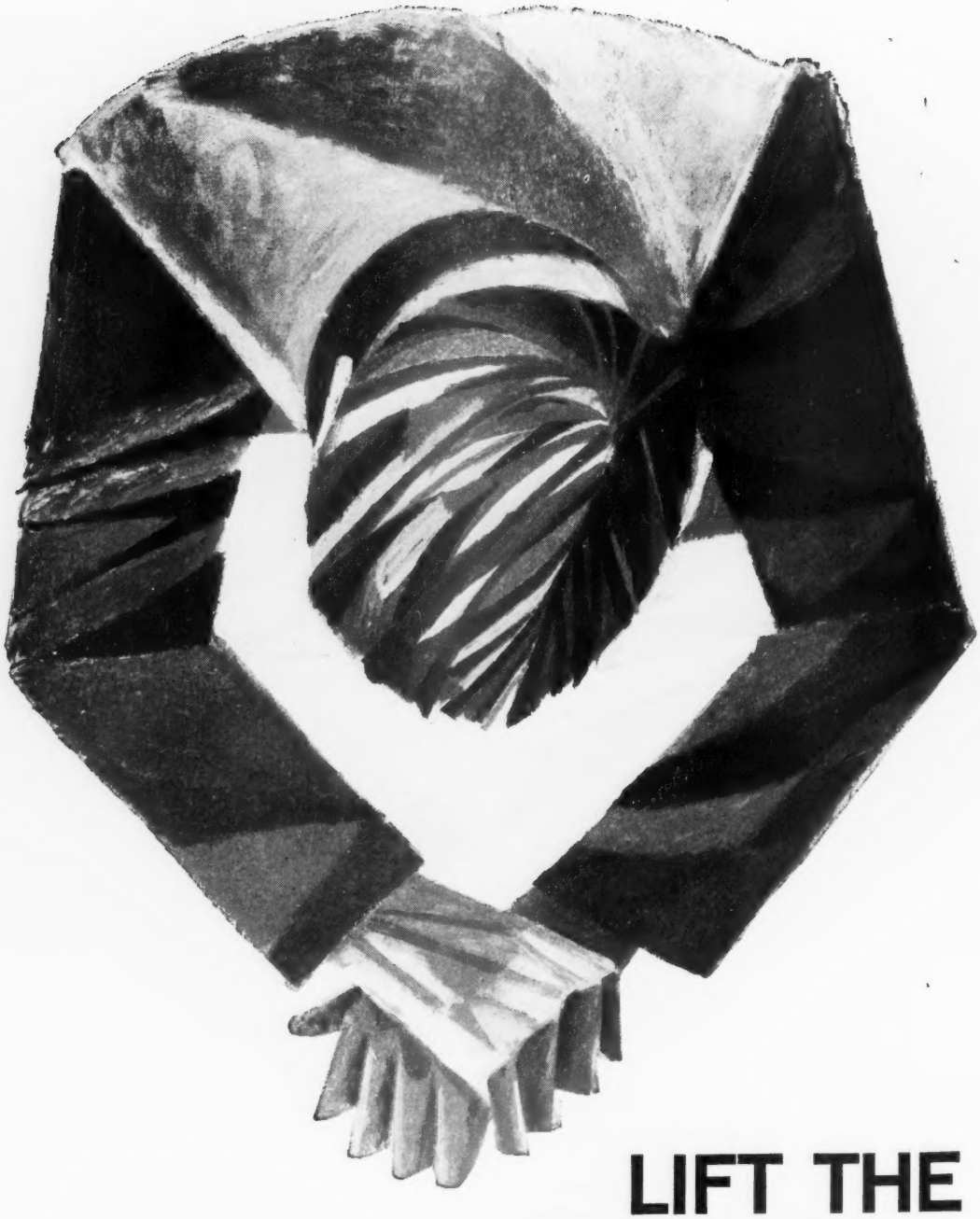

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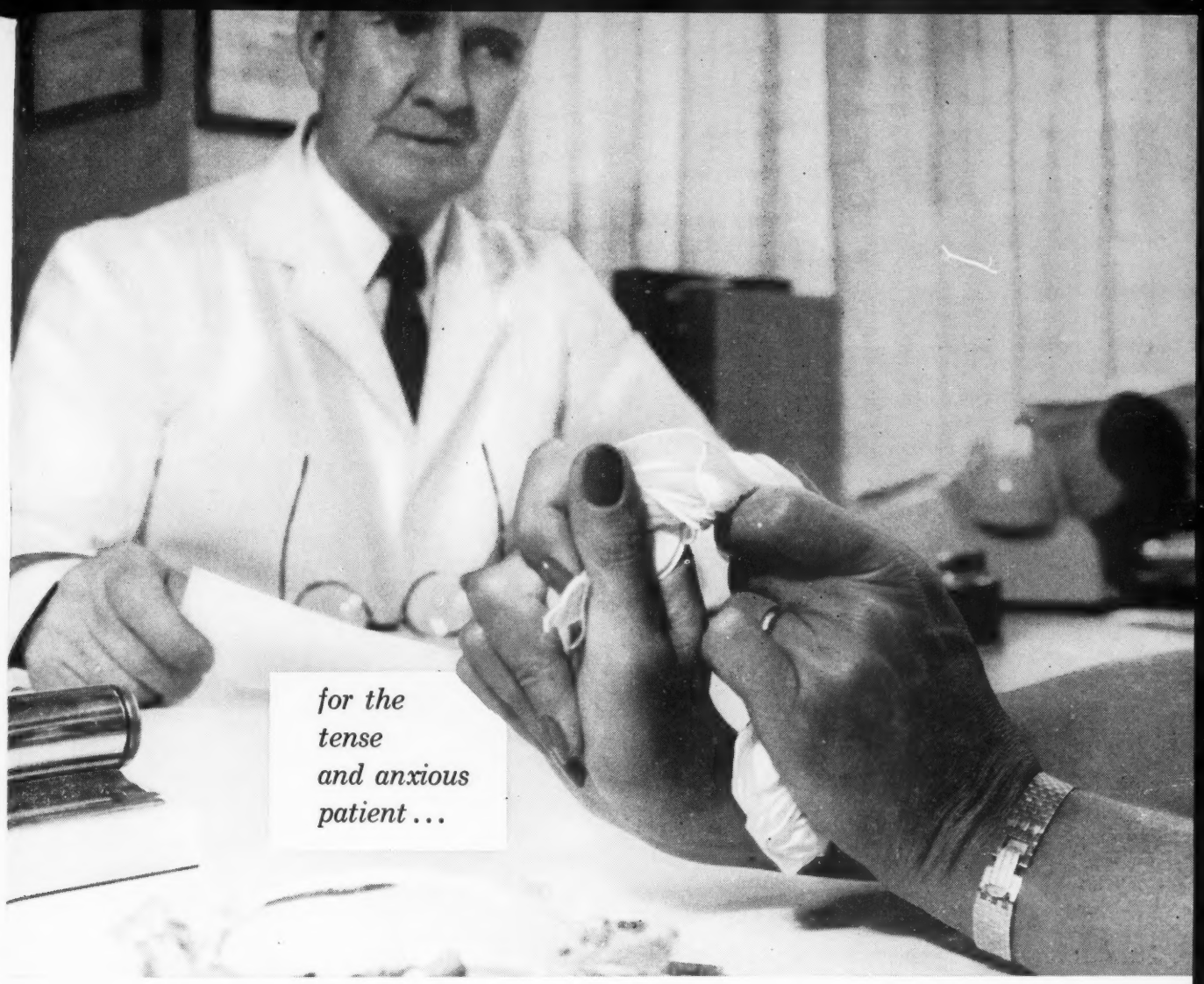

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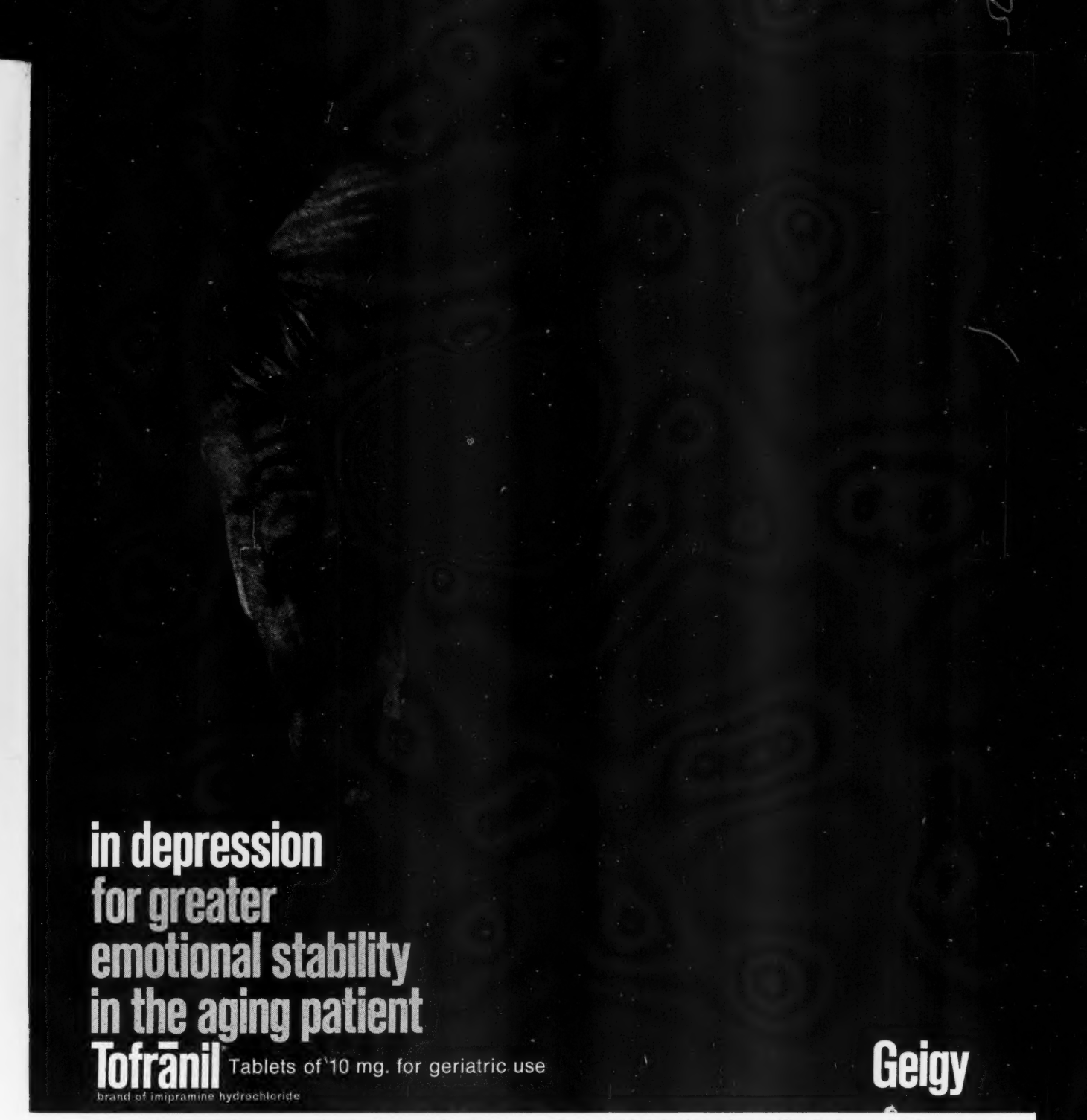

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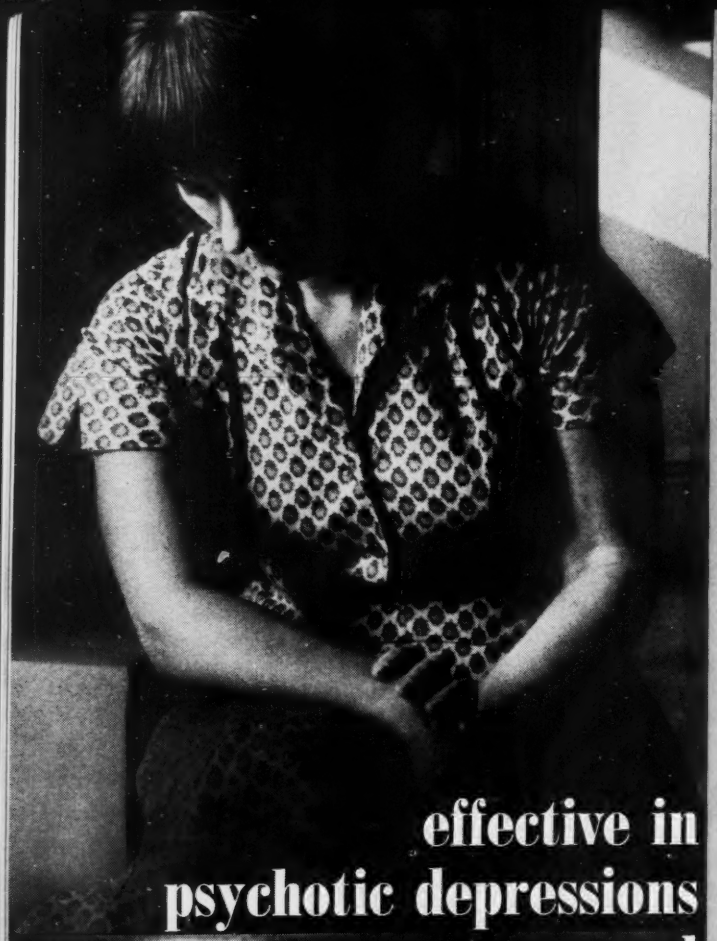

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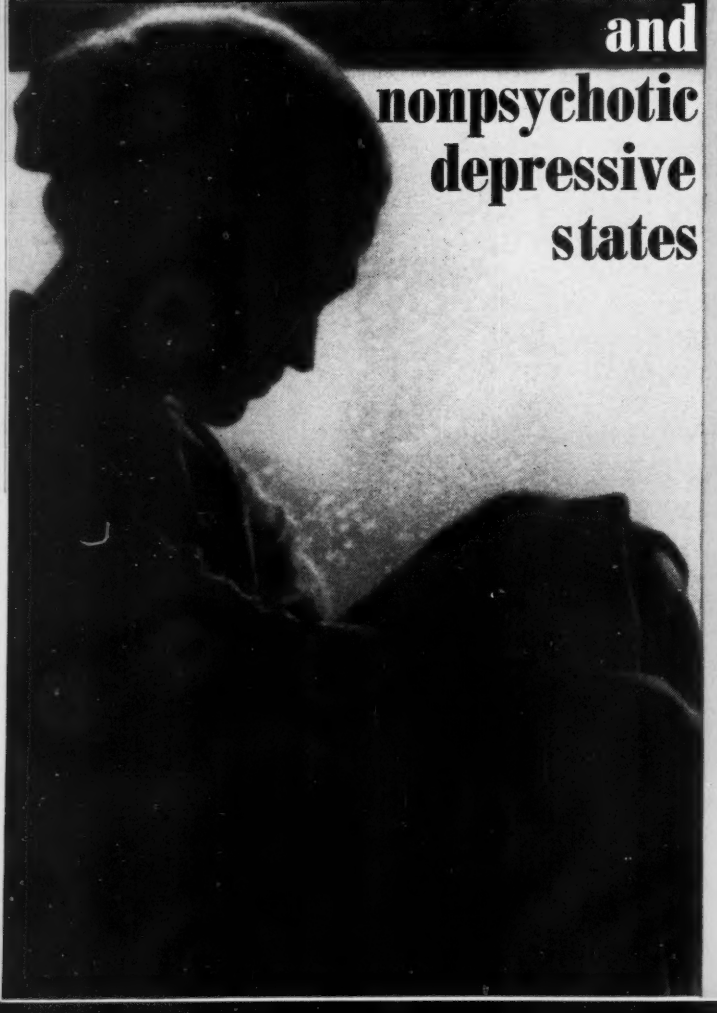

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DISEASES *of the* Nervous System

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Concurrent Paralysis Agitans and Schizophrenia

Confusion with Drug-Induced Extrapyramidal Disorders

LEO E. HOLLISTER, M.D., and FREDERICK S. GLAZENER, M.D.

Extrapyramidal syndromes are frequent during therapy of schizophrenic reactions with phenothiazine derivatives and Rauwolfia alkaloids.[1] As yet, no case of drug-induced paralysis agitans has become permanent. The coincidence of schizophrenia and spontaneously-occurring paralysis agitans could lead to mistakenly ascribing the latter to drug therapy. We decided to study the concurrence of these two disorders by reviewing cases of paralysis agitans admitted to a mental hospital. While doing so, we encountered a patient in whom this very problem arose.

Methods and Results of Study

All cases of paralysis agitans or Parkinson syndrome admitted during the period 1949 to 1959 were considered. Records were reviewed on 36 patients with unequivocal diagnoses to determine the frequency of associated psychiatric syndromes. Because this sample was recruited from a mental hospital, the frequency of associated mental syndromes was obviously not representative of a complete population of patients with paralysis agitans. In 31 patients there was an associated psychiatric diagnosis, eight being diagnosed as schizophrenic reactions. Five patients had no associated psychiatric diagnosis.

The eight patients with associated diagnoses of schizophrenic reactions were compared with the remaining 28 in respect to a number of clinical factors (Table 1). The major differences were the age of onset of mental symptoms (far younger in the schizophrenic group) and in the sequence of mental symptoms and paralysis agitans (most schizophrenics showing mental illness first, most others developing mental symptoms after manifest neurologic disease). The nature of the mental disturbance also differed, as might have been expected.

One of the patients with a schizophrenic

TABLE I

Clinical Manifestations of Paralysis Agitans With and Without Associated Schizophrenic Reaction

	Paralysis agitans with schizophrenia —8 cases	Paralysis agitans without schizophrenia —28 cases
Age at Onset of Paralysis Agitans	24–75 yrs.	26–74 yrs.
Post-encephalitic	4	21
Arteriosclerotic	1	5
Idiopathic	3	2
Age at Onset of Mental Illness	25–36 yrs.	32–72 yrs.
Schizophrenic react.	8	
CBS, Parkinson's dis.		10
CBS, senile/arterio.		5
Psychotic depression		4
Others		4
None		5
Clinical Manifestations of Mental Illness		
Delusions	+	+
Hallucinations	+	
Confusion	+	+
Disorientation		+
Memory loss		+
Loss insight	+	
Poor social adj.	+	
Depression		+
Emotional lability		+

From Veterans Administration Hospital, Palo Alto, Calif.

reaction was being treated with a phenothiazine tranquilizer as paralysis agitans was developing. The following case report indicates the confusion which ensued regarding the cause of his neurologic disease.

Case Report

A 48-year-old single Filippino man was admitted to Veterans Administration Hospital, Palo Alto, for the second time on 3/24/59 because of reactivation of a schizophrenic reaction.

Family history was negative for mental or neurologic disorders. He had lived in the United States since 1930, being in military service from 1942 to 1946. Soon after discharge from military service, he developed delusions of persecution, auditory hallucinations and a feeling of impending doom. He was committed to a California State Hospital where he received electroconvulsive therapy. Subsequently he was transferred to Veterans Administration Hospital, Palo Alto, being finally discharged in 1948.

His adjustment outside the hospital was marginal. He worked as a dish-washer and was living alone with little social contact. Shortly before his second admission he had been fired from his job and was found by his brother sitting in his room, hungry and destitute. He was sent to the hospital without any prior treatment.

At time of admission he was confused, disoriented and incapable of giving any coherent history. Casual mention was made of progressive muscular stiffness. He was a small, slow-moving and placid-faced patient. General physical examination was not remarkable. Neurological examination revealed marked muscle rigidity, which apparently could be relaxed voluntarily. No limitation of joint motion was noted. Plantar responses were flexor.

On 4/7/59, after being treated with 450 mg. of chlorpromazine daily for eight days, he was noted to have peculiar flexed, festinating gait. An extrapyramidal reaction associated with chlorpromazine administration was considered. Subsequently he was treated with prochlorperazine (4/21/59) in doses up to 75 mg. daily. Again he showed a stiff, festinating gait with frequent falls. Benztropine methanesulfonate (Cogentin) was added at a dosage of 4 mg. daily without apparent benefit. By 6/10/59, all drugs were discontinued. Temporarily he brightened and moved with assistance. His psychosis continued active.

Although tranquilizing drugs were avoided on the premise that they were causing extrapyramidal effects, generalized muscle rigidity, cog-wheeling, masked facies and contractures of the hands steadily developed over the next several months. Lumbar puncture revealed cerebrospinal fluid normal in appearance and under normal pressure; protein was mildly elevated to 54 mg. per 100 ml. X-rays of the skull were negative. Electroencephalogram showed a diffusely asynchronous pattern.

By 4/14/60, the patient had become helpless and bed-fast. His disease was steadily progressive from that time on, accompanied by failing nutrition. He died on 9/20/60. Post-mortem examination revealed findings compatible with a chronic meningo-encephalitis of unknown cause.

Comment: The psychotic symptoms on admission obscured his chief complaint of increasing muscle stiffness. As the clinical signs of extrapyramidal disease seemed to be aggravated by the phenothiazine tranquilizers, medication was discontinued on the assumption that the neurologic disorder would remit. Instead, it progressed. Retrospectively, it became obvious that the patient had progressive neurologic disease which preceded administration of any tranquilizing drug. Thus, a spontaneously-appearing paralysis agitans syndrome coincided with relapse of a schizophrenic reaction. Use of phenothiazine tranquilizers to treat the latter disorder led to confusion about the cause of the former.

Discussion

Almost 40 years ago, 34 percent of 146 cases of paralysis agitans were found to have mental symptoms.[2] The possibility of confusion in diagnosis when mental symptoms preceded the neurologic disease was noted in another report, which described emotional depression, agitation, sensory hallucinations, somatic or paranoid delusions, and memory defects as being prominent.[3] At least one of the five cases reported could have been regarded as having a schizophrenic-like picture preceding the neurologic disease. Confusion, emotional imbalance, delusions, dementia and auditory hallucinations were noted most frequently in another group of patients with post-encephalitic disorders.[4] Although one patient was listed as schizophrenic in another series of cases of paralysis agitans, review of these with onset under 50 years of age indicated that probably 10 of 72 would have been classified as schizophrenic reactions by present-day standards.[5] Some authors have questioned whether a specific psychosis is associated with Parkinson's disease. Four types of psychiatric syndromes have been proposed: 1) coincident unrelated psychiatric illness, 2) reactive mental disturbances, 3) psychiatric symp-

toms from medication (atropine group) and 4) paroxysmal psychiatric disorders (anxiety, depression, paranoid, schizoid, compulsive or agitative attacks) analogous to oculogyric crises.[6]

The present study indicated that a mental syndrome indistinguishable from schizophrenia may precede paralysis agitans by many years. If, as happened in the case reported, the neurologic symptoms become prominent during treatment with tranquilizing drugs, they might be erroneously attributed to the drugs. Progression of neurologic disease despite discontinuing drugs more likely represents concurrence of two disorders rather than irreversible drug effects.

Summary

Eight patients of 36 admitted to a mental hospital with paralysis agitans had concurrent schizophrenic reactions. Unlike other mental syndromes associated with paralysis agitans, schizophrenic reactions appeared earlier in life, usually preceding the neurologic disease.

One patient with schizophrenia of 12 years duration developed symptoms of paralysis agitans while under treatment with phenothiazine tranquilizers. The disease had actually begun before drug therapy was started, which became evident when the history was carefully considered. Discontinuation of medication failed to reverse the neurologic disease, as would have been expected with drug-induced extrapyramidal syndromes.

The possible chance association of these two disorders should be considered before any permanent extrapyramidal syndrome is attributed to tranquilizing drugs.

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Orthostatic Hypotension Secondary to Psychotropic Drugs

Treatment with Emphasis on Parenteral Ritalin

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Orthostatic hypotension is usually the result of a disease of the autonomic nervous system. There are numerous systemic conditions believed to be factors predisposing to postural hypotension. Old age, arteriosclerosis, CNS lues, hypertension, diabetes, endocrine deficiency, particularly involving the thyroid, adrenal and pituitary glands, febrile infections, blood dyscrasia, exhaustion states and cachexia, intoxication due to barbiturates, alcohol and other drugs are conditions where an imbalance of cardiovascular tone may result.

In 1959 over 20 million new prescriptions for tranquilizers were filled.[1] New psychotropic drugs are marketed usually emphasizing advantages, while side effects are minimized. Orthostatic hypotension is a complication not infrequently observed during the treatment with tranquilizers especially phenothiazine derivatives and some central nervous stimulants and antidepressants, mainly monoamine oxidase inhibitors. It occurs frequently as a side effect from potent antihypertensive medication, particularly rauwolfia products and ganglionic blocking agents. Without blaming the indiscriminate use of these drugs, it should be stressed that many physicians, the specialist as well as the general prac-

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titioner, do not always take into account the danger and complications due to postural hypotension. It is noteworthy that in the past hypotension was not even recognized as a disease entity. Treatment was rarely advised or indicated. Only with the advent of the psychotropic drugs has the incidence of hypotension become a major factor in treatment. This complication, insidious in its onset, is often overlooked and remains undiagnosed and untreated until a sudden circulatory vascular collapse in form of syncope occurs.

It is beyond the scope of this paper to discuss the intrinsic cause of orthostatic hypotension. Its intricate mechanism of onset is poorly understood. Venous pooling seems to play an important role. Some hypotheses deal with the influence of the monoamines on the action of serotonin and epinephrine. Page[2] has stressed first, that serotonin has a strong direct vasoconstrictor action; second, that abolition of neurogenic vasomotor tone is one of the most important determinants of the blood pressure response to serotonin. When nervous tone is high, the response is predominantly hypotensive and when low, hypertensive. These factors may be influential in the event of abnormal blood pressure responses to some drugs. It is significant that only a minor number of patients will show clinical symptoms due to orthostatic hypotension. It may be presumed that physiological defense mechanisms responsible for a normal homeostatic balance are impaired due to underlying predisposing conditions. One of the reasons that hypotension is overlooked is its insidious onset. It must be kept in mind that it may occur at any time during treatment. Only constant observation of the patient with frequent blood pressure readings, particularly upon arising, will reveal an early diagnosis. Symptoms leading to the diagnosis of postural hypotension are vertigo, fainting or dizzy spells, confusion, weakness, malaise, weight loss, anorexia and insomnia, when found during treatment with phenothiazine derivatives, MAO inhibitors or antihypertensive drugs.

Clinically three major sequelae of hypotension deserve special consideration and discussion: 1. Coronary occlusion, 2. Shock, 3. Cerebrovascular insufficiency.

Myocardial infarction and coronary occlusion occur as a rare incidence during treatment with phenothiazine derivatives. In narrowed vessels, predisposed by arteriosclerotic, luetic, or other vascular changes, a transient hypotension may cause a definite decrease in blood flow, particularly through the coronaries and precipitate a thrombotic process which may result in myocardial infarction or total occlusion with sudden death. A similar sequence of events is often postulated at autopsy as the cause of the coronary occlusion. It is possible that there are many cases in which a relation between sudden death and tranquilizing medication has existed but has not been correlated.

Recently the following case was observed:

A 46-year-old white male hospitalized for three years with a diagnosis of paranoid schizophrenia, complained early in the morning of severe pain "all over" becoming extremely agitated and restless. He had been on imipramine (Tofranil b.i.d.) for several months. He was given chlorpromazine 50 mg. i.m. at 8:00 A.M. Blood pressure before and after the injection was normal. He quieted down for a while but shortly after noon he again became severely disturbed. He was given a second injection of 50 mg. chlorpromazine i.m. exactly four and one-half hours after the first treatment. He immediately complained of feeling weak. Blood pressure dropped suddenly, he became unconscious and five minutes after the injection respiration ceased and he was pronounced dead. Autopsy revealed an acute thrombosis of the anterior coronary artery, no other lesions. The past history revealed that three years prior to his death he had an abnormal electrocardiogram indicative of coronary artery disease. EKG's repeated one and three months later were normal. There was no physical complaint until his death.

If postural hypotension persists for some time without treatment the condition may become irreversible; this means hypotension will be found in either supine or vertical position. If blood pressure levels drop below 80 mm. Hg. systolic a condition of shock may ensue. Sudden onset will be evidenced by syncope. If onset is slow it may remain unnoticed and treatment may be given too late.

The following case illustrates such a situation:

A 19-year-old, obese white girl hospitalized three years before with a schizophrenic reaction, hebephrenic type, and since had been irritable and of difficult management was readmitted five days prior to her death with an acute febrile condition, temperatures ranging from 104° to 105.5°. Blood pressure varied from 240/140 to 180/110. There was no previous history of hypertension. She was treated with combined antibiotics (streptomycin plus penicillin). Temperature returned to normal within four days, blood pressure also returned to normal levels averaging 130/85. On her fifth hospital day she was doing well physically but became mentally very disturbed. Because of threatening violent behavior 50 mg. of promazine i.m. was given at 4 P.M. She became quieted and remained in bed. A few hours later she became motionless; her color changed to a grayish paleness. Blood pressure dropped to 100/60 and later 88/60. She was found to have no major complaints and was not believed to be critically ill. She expired at 2 A.M., ten hours following the injection of promazine. Autopsy was refused. No diagnosis was made as to the acute febrile illness. One has to assume that this patient went into irreversible shock which was not diagnosed in time for adequate treatment. One may also speculate that there was a latent cardiovascular instability, possibly contingent to the acute febrile illness, predisposing to the onset of a hypotensive episode.

Cerebrovascular insufficiency caused by hypotension is a problem in the elderly patient. The hazards of tranquilizing drugs in the treatment of the senile have been reported for some time.[3] It has been stressed that irritability, confusion and deterioration has often followed the use of hypotensive drugs. This may be explained by the possibility of severe brain damage resulting from even a mild hypotensive episode. Again it has to be stated that preexisting vascular changes predispose to complications ranging from a major hemorrhage or thrombosis to multiple small scattered lesions sufficient to cause severe functional loss. Therefore, one must caution against the indiscriminate use of hypotensive tranquilizers and MAO inhibitors in the senile.

In the past the treatment of hypotension was entirely symptomatic with rest in the recumbent or slight Trendelenburg position and the administration of vasopressor drugs like caffeine, benzedrine, ephedrine, adrenalin and amphetamines. It has been noted recently that some of these pressor agents fail to restore the vasomotor tone in hypotension caused by psychotropic drugs, particularly phenothiazine derivatives. It was frequently found that these compounds would reverse the usual elevating action of the pressor agents, particularly epinephrine, and cause a further lowering of blood pressure. The mechanism of this reversal of pharmacotropic action is unknown. It is now accepted practice to restore vasomotor stability with intravenous infusion of Levophed, (levarterenol, 4 cc. in 1000 cc. of 5% glucose solution) or intramuscular Neosynephrine (phenylephrine 1 to 10 mgs.).

In a recent study[4] of the effects of parenteral Ritalin (CIBA Pharm.—methylphenidate), a cerebral stimulant, it was found that this drug has a definite vasopressor effect and could be useful in the treatment of postural hypotension secondary to psychotropic drugs. Ritalin can be safely given with most phenothiazines. Its preventive use in doses of 2 to 4 cc. i.v. (individual sensitivity may warrant the use of 1 to 2 cc. only) makes the parenteral use of phenothiazines such as chlorpromazine, promazine, perphenazine and others safer, preventing a sudden dangerous drop of blood pressure to shock level. This is particularly recommended in known cases of a labile cardiovascular system. In severe hypotension with blood pressure close to shock level intravenous Ritalin is indicated; in milder cases intramuscular use of 2 to 4 cc. of the drug may be sufficient. Orthostatic hypotension secondary to psychotropic drugs will not always disappear with discontinuation of the causative agent. At times a period of several months is needed to control the condition. This happens more frequently in the senile or debilitated patient. It was found that Ritalin is especially helpful when prolonged treatment is required. It seems that Ritalin has a "tonic" effect on the vasomotor tone and therefore is of more lasting therapeutic value than other vasopressor agents.

When hypotension occurs with symptoms of anorexia, weight loss and weakness, an invigorating dietary regimen is imperative with high calory intake, multiple vitamins

and eventual stmulation with insulin (5 U regular before meals).

While intravenous Levophed is doubtless a life saving medication in severe shock, intravenous Ritalin has a very similar action. It has the advantage of not requiring an infusion set. It is superior to intramuscular Neosynephrine. Both medications were given to 10 hypotensive patients comparing 0.5 cc. of Neosynephrine i.m. to 2 cc. Ritalin i.v. and i.m. The results showed that Ritalin had a stronger vasopressor effect. Side reactions[1] of Ritalin when administered in hypotensive cases are minimal if the lowest therapeutic dose is given. Initial test doses should not exceed 2 cc. i.v. or i.m.

The following two case reports illustrate the treatment of secondary hypotension with parenteral Ritalin.

Case No. 1. A 67-year-old white man, institutionalized 40 years with diagnosis of paranoid schizophrenia, had episodes of aggressive and destructive behavior off and on during his entire hospitalization. Therapy with trifluoperazine began early in 1959 without improvement. Symptoms of cerebrovascular insufficiency appeared and he became confused and depressed, had weak, dizzy and fainting spells, severe anorexia and lost over 30 pounds. Finally he was discovered to have hypotension which was particularly severe upon arising, with blood pressure levels varying from 75/40 to 85/50. He was given intravenous Ritalin, 2 cc. daily. At first only transitory improvement was noted; subsequently his general condition improved when placed on a vigorous dietary regimen and given insulin 5 U regular before meals. The hypotension persisted for several months with readings below 90 systolic in the morning and 95 to 110 later in the day. Intravenous Ritalin was continued for two weeks on a daily basis; later it was given intramuscularly two or three times weekly until the vascular tone was sufficiently re-established and treatment could be discontinued. It may be stressed that the antidepressant stimulant action of parenteral Ritalin was of great importance in the over-all improvement of the patient.

Case No. 2. A 31-year-old white man, hospitalized six months for schizophrenia did not respond to medication with various tranquilizers, particularly perphenazine, chlorpromazine and trifluoperazine in average doses. He had episodes of severe confusion, appeared to be organically ill with marked pallor, weight loss and staggering gait. It was finally found that patient was affected by postural hypotension. The diagnosis was made when patient suffered a sudden syncope following an injection of chlorpromazine 50 mg. i.m. He remained unconscious for several minutes, blood pressure dropped to nearly shock level (systolic below 70). A similar episode occurred two days later following an injection of chlorpromazine 25 mg. i.m. In both instances he was treated with analeptics. The hypotension was manifested with systolic blood pressure levels around 80 to 85 upon arising not exceeding 110 during the rest of the day. There was a potential predisposition to cardiovascular lability; several years before he had had his left kidney removed due to a congenital cystic condition. It was believed that this factor could be contributory to the onset of hypotension.

He was treated with Ritalin 2 cc. i.v. for several days. Therapy was continued with intramuscular injections two to three times a week until the general cardiovascular motor tone improved.

Summary

Orthostatic hypotension secondary to psychotropic drugs is a side effect often underestimated by medical investigators. The mechanism of onset is poorly understood. Its major complications are 1) coronary occlusion (myocardial infarction); 2) shock; 3) cerebrovascular insufficiency. The treatment discussed emphasizes the use of parenteral Ritalin.

Author's Note: The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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Regressive Convulsive Therapy and Lobotomy in the Treatment of Mental Disorders

JAMES L. SAGEBIEL, M.D.

This report deals with 267 cases of schizophrenia given various degrees of regressive and non-regressive electroshock therapy (EST) from 1946 to 1960. Of these, 19 ultimately came to lobotomy and are included in that group (Table III). In addition, there were 20 non-schizophrenic patients who were lobotomized by the Grantham technic after insulin or electroshock therapy had failed. One schizophrenic was lobotomized without previous EST. Only cases of schizophrenia were selected for the regressive EST study because of their well-known refractoriness to all kinds of therapy.

It may be significant that these 267 schizophrenics, picked out of approximately 1200 cases of all types given EST, were from private practice. Thus there has been a gradual evolution of my thinking with increasing confidence in the use of deliberate regressive EST. In a large institution one can select a "block" of cases on which to do a deliberate experiment in the field of regressive EST or of chemotherapy. Since the cases presented here are all private cases certain problems and limitations arise; for example, (1) pleasing the relatives and allaying their anxieties, (2) discontinuation of the treatment due to fear of it, (3) limitation of finances which might cause them to seek discharge from the hospital or transfer to a state hospital, (4) inability to follow them up for as long as one would wish. After the patient becomes symptom free through EST, he returns home and goes through the various phases of rehabilitation with adjustment to the home, to the community and finally to the work environment. He may be soon lost track of because he does not wish to incur additional expense when he is feeling well. Thus in private practice we are in a position which requires that we must assume the patient is doing well unless he returns for further therapy.

The clinical material showed a male to female relationship of 1-1.4; average age was 36 years with a range of 14 to 68 years; the average number of EST's was 42 ranging from 8 to 370. The 370 treatments were given over many years with frequent intervals of observation and office therapy.

The patient was prepared for treatment by the intravenous injection of 0.25 g. of Sodium Pentothal and 1 mg. of atropine, followed in another syringe by 10 mg. succinylcholine chloride (Anectine). Bitemporal electrodes were applied and the patient was given 2 to 4 grand mal convulsions by alternating current at two minute intervals, daily, six days a week. Rarely he would receive 5 to 6 convulsions daily for a short period to overcome anxiety or disturbing symptoms. Toward the end of the course the convulsions were reduced to one daily, then every other day.

The results were classified as "improved" when the patient was symptom free and returned to his former socio-economic status or when he retained residual symptoms but was not disabled for limited social or economic activity. They were classed as "poor" when there was no improvement.

It was found desirable to divide these cases into three chronological periods of treatment:

(1) *From 1946 to 1952* regression was inadvertent or accidental. This could be described as the period in which *mild* regression was used. Even then I found that in 82 cases, 71% of 17 cases improved when regressed, whereas 49% of 65 non-regressed patients showed poor results (Table I).

This seemed to indicate that regression, even though inadvertent, represented a faforable factor and that lack of regression was a poor prognostic sign. When it became

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evident that more than 50% of these schizophrenics did not get well when subjected to mild or orthodox EST, there began a trend toward more intensive treatment using two or occasionally three convulsions daily.

At this time the question arose as to whether brain damage was incurred by this method. Therefore a series of 125 consecutive cases were subjected to spinal fluid examinations before and after treatment. In those requiring a long course of treatment the spinal fluid was checked during its course. In no case was any deviation from normal found. This also applied to blood pressure, complete blood count, fasting blood sugar and urea nitrogen. Many elevated blood pressures returned to normal during treatment only to rise again when treatment was stopped. Occasionally there was a mild fever of 100°-101°F. Isolated spikes caused no concern but if the elevation persisted, EST would be stopped while a search for upper respiratory infection, pulmonary or genito-urinary pathology was made. If no pathology could be demonstrated, EST was resumed after the temperature remained normal for 48 hours.

(2) *In the period from 1952-1956,* which might be called a transitional period in my trend toward deliberate regressive EST for schizophrenics, it was found that in 45 cases, 100% of eight cases improved when regressed, whereas 50% of 37 non-regressed patients obtained poor results (Table I). This appeared to be still more confirmatory evidence in favor of intensive EST for schizophrenic patients.

(3) *From 1956-1960,* therefore, I deliberately attempted to regress *all* schizophrenics. In this group of 121 cases, subjected to regressive EST 92% of 65 cases improved when regressed and 36% of 56 cases obtained poor results when they failed to regress (Table I). By this time I was using mostly three or four convulsions daily at two minute intervals. The totals of these three chronological periods are summarized in Table II.

After failure of regressive EST 19 additional cases were lobotomized by the Grantham technic (Table III).

Thus, in the total series of 267 schizophrenic patients, 84% of 95 cases improved when they regressed and 49% of 172 cases had poor results when they did not regress (Table IV). This indicates that regression in the treatment of schizophrenia is of value and that failure to regress during EST is a poor prognostic sign.

This matter of regressive EST is not new. In 1948 Kennedy and Anshell[1] reported on a number of patients whom they regressed to the infantile state and 66% of these went into remission. They concluded that regression was beneficial.

Cerletti[2] in August 1950 reported that multiple EST's seemed to have a good influence in obsessive states, psychogenic depressions and even in some paranoid cases. He labeled this type of therapy "annihilation technic." This annihilation syndrome has been compared by Cerquetelli and Catallano with

TABLE I

Schizophrenics Treated with Regressive EST

		Regressed		Non-Regressed	
1946-1952 *Types*		Improved	Poor	Improved	Poor
Paranoid	(52)	8	3	20	21
Catatonic	(14)	2	1	7	4
Affective	(4)	0	0	2	2
Simple	(4)	1	1	2	0
Miscellaneous	(8)	1	0	2	5
	82	12	5	33	32
		71%	19%	51%	49%
1952-1955					
Paranoid	(35)	7	0	13	15
Catatonic	(2)	0	0	0	2
Affective	(3)	1	0	2	0
Simple	(1)	0	0	1	0
Miscellaneous	(4)	0	0	2	2
	45	8	0	18	19
		100%	0%	50%	50%
1956-1960					
Paranoid	(69)	34	5	19	11
Catatonic	(18)	6	0	8	4
Affective	(16)	10	0	3	3
Simple	(7)	4	0	2	1
Miscellaneous	(11)	6	0	4	1
	121	60	5	36	20
		92%	8%	64%	36%

the psychopathology following prefrontal lobotomy. They felt that there was a close parallel, with the advantage of reversibility in case of shock. Cerletti for the first time remarked on the emergence of anxiety during EST.

Garrett and Mockbee[3] in 1952 treated a series of 30 patients with 30 controls by regressive therapy. They reduced them to an "organic syndrome" but found that on emergence from this there were no ill effects from intensive regressive electroshock. Observations by electroencephalography revealed little change except for high voltage and slow dysrhythmia, most prominent in the anterior part of the brain, which lasted about 14 days after the termination of shock. The rhythm then became fairly normal. Some irregularity was noted for two or three months after the termination of treatment.

By 1957 many investigators were seriously engaging in the study of regressive EST. Among them were Glueck, Reiss and Bernard[4] who reported on a series of 100 cases, 67% of whom were schizophrenics with previous adequate courses of EST and/or insulin coma therapy without lasting improvement; 33% had had no treatment.

Glueck remarked that many of the investigators who were unsure of the margin of safety were restricted by the fear of inducing irreparable organic damage; thus they limited the number of treatments even though many patients were not completely regressed according to later standards. He felt that results in this group might have been improved if they had used more intensive therapy. Glueck described the effects of regressive shock therapy along both mental and neurological lines. He noted especially that at times a small number reached a plateau where, with continued EST, instead of regressing, they became more alert in spite of continued treatment. Calloway and Boucher[5] in 1950 concluded, in their EEG studies on a series of cases intensively treated, that EST produced no more permanent EEG changes than orthodox EST. Glueck confirmed these findings. He felt that regressive electroshock therapy proved its merit in chronic schizophrenics refractory to previous adequate courses of EST and/or insulin. He believed regressive shock therapy should be tried as the treatment of choice when hospitalization was necessary in schizophrenics. Since it does not result in cerebral impairment and final results are far superior to standard forms of EST, Glueck felt it should supplant the standard form even in patients who seemingly had a good

TABLE II

Schizophrenics Treated with Regressive EST

1946-1960

			Regressed		Non-Regressed	
Types			Improved	Poor	Improved	Poor
Paranoid		156	49	8	52	47
Catatonic		34	8	1	15	10
Schizo-Affective		23	11	0	7	5
Simple		12	5	1	5	1
Miscellaneous		23				
Pseudo-Neurotic	(8)		1	0	4	3
Chr. Undiff.	(6)		3	0	2	1
Hebephrenic	(5)		1	0	0	4
Over-Ideational	(3)		1	0	2	0
Post-Traumatic	(1)		1	0	0	0
		248	80	10	87	71
			89%	11%	55%	45%

TABLE III

EST Effects on Pre-Lobotomized Schizophrenics

		Regressed		Non-Regressed	
Types		Improved	Poor	Improved	Poor
Paranoid	(10)	0	3	0	7
Catatonic	(2)	0	1	0	1
Schizo-Affective	(6)	0	1	0	5
Pseudo-Neurotic	(1)	0	0	0	1
	19	0	5	0	14
		0%	100%	0%	100%

TABLE IV

Total Schizophrenics

	Regressed		Non-Regressed	
	Improved	Poor	Improved	Poor
267	80 (84%)	15 (16%)	88 (51%)	84 (49%)

prognosis as they only too often would relapse in a few months. Regressive electroshock therapy should be given before any patient is subjected to psychosurgery.

In August 1958 Moore[6] reported that regressive electroshock therapy was helpful in many cases of schizophrenia. His theory was that if sufficient EST was given so that the patient remained in a state of confusion for a lengthy period, then the "rest" from delusions and hallucinations might assist in recovery. It was an attempt to achieve a principle frequently used in the treatment of organic disease of the body, i.e. putting the diseased or injured parts at rest.

Cameron and Pande[7] reported on a new method of treatment of chronic paranoid schizophrenics in 1958. Their treatment consisted of prolonged sleep lasting 30 to 60 days combined with intensive EST. Sleep was produced by means of chlorpromazine, secobarbital, pentobarbital or phenobarbital, associated with EST given once daily or as many as four or five shocks within two or three minutes. The rate of administration of EST was set so that complete "depatterning" was achieved somewhere between the 30th and 60th day of sleep, usually after about 30 EST's. Thereafter, the amount of sleep-producing medication was gradually reduced as was the administration of EST. The period of rehabilitation following this type of treatment paralleled my own. These authors found chemical and physical therapies to be the only satisfactory means of halting the schizophrenic process. The objectives of intensive physical and chemical therapy were: (1) to bring the schizophrenic process to an end and (2) to break up completely, through the process of "depatterning," the ongoing structure of the abnormal behavioral patterns of the individual. This is similar to regressive EST.

It has become apparent in the past decade that the patient must pass through certain phases during EST in order to have the best chance of regaining a clinical status of normality.

The first phase noted was the remission phase. It was found during the first six or eight treatments in the average case that the patient lost the symptoms of which he complained on admission to the hospital. In prior years it was customary to dismiss the pa-

TABLE V
Results of Lobotomies

1954-1960			Immediate		Average	Final Results	
SCHIZOPHRENICS	(20)		Improved	Poor	Follow-up Time	Improved	Poor
Paranoid	(11)		10	1	All	10	1 d. 4 yr.
Catatonic	(2)		2	0	Schiz.	2	0
Affective	(6)		6	0	29 months	5	1
Pseudo-Neurotic	(1)		0	1		0	1
		20	18 (90%)	2 (10%)		17 (85%)	3 (15%)
NON-SCHIZOPHRENICS	(20)						
Manic-Depressive Psychosis	(5)		4	1	40 months	4	1
Invol. Psychosis	(6)		6	0	18 months	4	(2 Died)
Depr. Reaction	(2)		2	0	8 months	2	0
Psychoneuroses	(7)						
Phobic Reactions		(3)	3	0	All Psych.	3	0
Obsess.-Comp. N.		(3)	3	0	43 months	3	0
Conv. Hysteria		(1)	1	0		0	1
Total		20	19 (95%)	1 (5%)		16 (80%)	4 (20%)
		40	37 (93%)	3 (7%)	28 months average follow-up	33 (83%)	7 (17%)

tient from the hospital at this time thinking he was well. Too many recurrences appeared so that it was eventually decided this was only a preliminary phase. In this remission phase the patient unconsciously indicated that he would be willing to relinquish his psychotic symptoms and behave as an adult if the doctor would only stop the treatments.

It soon became evident that there was a *secondary phase called the anxiety phase.* In this, as the original symptoms disappeared, anxiety appeared. This took many forms, such as the patient pleading to go home, or finding excuses why he should go home with negative attitudes toward the treatment. This might progress all the way to violent resistance to taking the treatment. Many times the paient would frankly admit a fear of EST. In this phase we apparently mobilized the patient's unconscious anxieties which were at the basis of his psychosis. These anxieties would then "boil up" to the surface and become evident. This phase could be shortened by increasing the intensity of the treatment, i.e. by giving multiple EST's daily.

There is considerable overlapping of these various phases. The remission phase might continue throughout the entire treatment course, i.e. the patient would not get rid of his original symptoms. When this occurred it was a poor prognostic indicator. There might be multiple anxiety phases: primary, secondary or tertiary. The regression phase might be replaced by a reanimation phase with later regression.

As an overlapping phenomenon to the anxiety phase, however, *regression would routinely occur.* The patient would become progressively retarded, less communicative with others and would develop amnesia. With daily therapy this would continue until the patient was in an infantile state, unable to care for himself, requiring total care for feeding, bathing, clothing and bedding. He not infrequently would become incontinent of urine and feces. In this phase it seemed as if we returned him to an infantile state psychologically. This phase is usually quite prolonged. It took many years to realize that the patient would eventually emerge into a reanimation phase.

In the reanimation phase the patient would again begin to take an interest in his environment, feeding himself, taking care of his toilet and generally growing up. When these clinical phenomena appeared electroshock was diminished, tapered off and terminated.

The next phase is the reintegration phase. After EST was stopped the patient rapidly "grew up" from the infantile through the adolescent to the mature adult, resuming self care and regaining orientation. He regained contact with his environment, talked more and eventually lost his amnesia and confusion.

He then passed into a rehabilitation phase. When the above clinical phenomena appeared it was obvious that the patient had overcome his "psychological death." Seven to 10 days after termination of treatment the patient usually had regained his orientation; the confusion had disappeared and he was able to communicate, engage in occupational and and social therapy and take care of himself in a normal way. If reorientation did not occur or if confusion persisted with amnesia or with any of his former symptoms, it was obvious the treatment had not been successful. This was an indication for resumption of treatment.

In a successful case the patient would leave behind all his psychotic symptoms, the affect having been gradually "chipped off" from the psychotic constellation and externalized. After personal orientation occurred there was acceptance of those about him with social reorientation. Subsequently, there would occur adjustment of the individual to the hospital environment, to the home environment, then to the community environment and finally to his former employment.

Thus it can be seen that the primary anxiety phase must be considered as only one phase through which the individual goes in order to regain emotional stability and freedom from his psychotic symptoms.

Passing through these phases does not necessarily lead to a cure. Schizophrenia is such a complex disease with so many etiologic factors that no one method of treat-

ment can be certain to effect a cure. Nevertheless, regression therapy, followed through the various phases, seems to offer the best results so far.

Comment

It has been observed that many cases would first regress, then regain their symptoms, but then the symptoms might be of a different type; for example, an original paranoid schizophrenic might regress and then reanimate and develop catatonic symptoms to the point of catalepsy. This would indicate that the underlying disease was still present. When such a condition occurred it was a sign to the operator that the prognosis was poor and that the patient might not eventually regain a normal clinical status. Many of these cases, reviewed in retrospect, suggest that the patients should have had much more and heavier EST. Then, with prolonged occupational and social therapy, they would regain a normal mental status.

Many times it was noted that regressive electroshock therapy would bring out a latent paranoid, catatonic or hebephrenic type of ideation and behavior which had been covered by the ego defenses present at the initial investigation. It has been found that resistance to the standard dose of Sodium Pentothal or motor hyperactivity post-EST may be a measure of the underlying tensions, hostilities or refractoriness of the illness. It must be pointed out that in many cases it is difficult to differentiate between regressive symptoms and those representing an underlying catatonia. Many times there is an admixture of the two.

Azacyclonol (Frenquel) post-EST seems to be helpful in regaining contact with reality. At times some of the phenothiazines are useful in the post-EST period. These, however, are not considered curative.

Three deaths occurred which were directly related to EST. All were paranoid schizophrenics, two of whom had been alcoholics and one who gave a vague history of head injury 3 months before onset of his illness.

W.F.: Male, aged 49, an alcoholic for four years with a diagnosis of schizophrenia, paranoid type, received 64 EST's of a regressive type in divided courses between June and September 1950. He showed a consistent tendency to relapse and was eventually transferred to Dayton State Hospital where he died, three weeks after his last treatment. The autopsy showed: (1) Recent pulmonary infarction, (2) Acute exacerbation of chronic congestion of all organs, (3) Generalized arteriosclerosis, mild. Microscopic examination of the brain showed early encephalomalacia due to embolus.

P.M.: Male, aged 39, had a diagnosis of schizophrenia, paranoid type, with a vague history of head injury three months prior to admission. After this personality changes occurred. On a previous admission to the hospital he had received 17 ECT's. During this time his temperature had been above 100° sixteen times and above 102° twice. There was no known reason for this.

In June 1954 he was re-admitted to Miami Valley Hospital. Two days later a lumbar puncture was done which showed CSF normal. He received three EST's, ran a temperature to 103.2°, after which the treatments were stopped. Six days later he had a spontaneous convulsion and died.

Autopsy showed: (1) Bilateral pulmonary embolism with infarction, (2) Cerebral congestion well marked with early glial degeneration, (3) Enlargement of the hypophysis (1.5 times normal size).

H.H.: Male, aged 52, a paranoid schizophrenic of three years' duration gave a long history of alcoholism, had 25 EST's during March and April 1959. Soon after his last EST he developed peripheral vascular collapse probably secondary to factors other than cardiac origin. He developed an oliguria, his urea nitrogen which had been normal on admission rose to 126 mg.% and he became hypotensive. He died four days later.

The autopsy findings were: (1) Bronchopneumonia, bilaterally, (2) Chronic pyelonephritis, (3) Organized myocardial infarction and (4) Atherosclerosis of the aorta, advanced.

From these findings it is obvious that patients with a previous history of alcoholism, which is common in the paranoid schizophrenic, or those with a history of head injury should not be given regressive EST.

Lobotomies

Twenty schizophrenics were lobotomized, 19 of whom had had previous EST or insulin therapy with poor results. One patient, a chronic paranoid schizophrenic was of such long duration that it was felt a lobotomy was indicated without a test of shock therapy. There were 20 other cases lobotomized, as follows: Five manic-depressive psychoses, four of them in the depressed phase; six in-

volutional psychoses, five of them in the depressive phase, the other a paranoid; two depressive reactions, one complicated by alcoholism; three phobic reactions; three obsessive-compulsive neuroses; and one conversion hysteria.

The technic used was that devised by Grantham[8] of Louisville, Ky., who electrocoagulated the ventromedial quadrant of both frontal lobes, assuming that most of the affective fibers passed from the thalamus to this area. Apparently this assumption was correct as the procedure does remove the *overload* of affect bombarding the frontal lobes. The criteria established for the selection of cases for lobotomy was overt anxiety or hostility or any of its many symptomatic manifestations.[9]

Two patients died in this series of 40 lobotomies. A 74-year-old male with diabetes who had an involutional psychosis with agitated depression died one month after his second lobotomy dut to failure to adhere to his diabetic program. The other was an involutional psychosis of a depressive type in a 57-year-old male who had had two cerebral thromboses seven and four months previously. Hypertension had been known for three years. He had not had previous EST. He had a good immediate reaction to the operation but died 12 days later of a coronary attack.

There were three complications, one severe and two mild. The mild ones consisted of post-operative major motor convulsions which were easily controlled by anticonvulsant medication. The severe complication was in a patient with an obsessive-compulsive neurosis, a male aged 57, who had not had previous EST because of a retinal detachment. After having a second lobotomy he was in good spirits and free of his anxieties and compulsions for 24 hours. He then had a grand mal seizure after which he was in a confused restless state. He was thought to have had a left parietal thrombosis. Within the next six hours he had 10 grand mal convulsions and was semi-comatose. The following day he was confused, non-responsive and did not recognize his relatives. He repeated words or phrases without comprehension. This continued for about four weeks with gradual improvement but it required seven months for him to fully regain his reading ability. During this time he had two grand mal convulsions in spite of anticonvulsant therapy. During the past three years, however, he has been working periodically. The electroencephalogram has been normal. Seven months after his second lobotomy he was reading with correct understanding of the content. He has had no convulsions for one year but is still under anticonvulsant medication. His compulsive activities have been minimal without anxiety.

Results of Grantham Lobotomies (Table V)

In the 20 schizophrenics lobotomized by the Grantham technic, 18 (90%) improved immediately after operation while 2 (10%) had poor results. After an average of 29 months observation, 17 (85%) were improved, while 3 (15%) were classified as poor results (one died).

The five manic-depressive psychoses did fairly well; four improved and the other had a poor result. The same appeared in the final results after 40 months average observation.

All six involutional psychoses had good immediate results. In the final results, after 18 months average observation, four were improved. Two died and are mentioned under deaths in Table V.

The depressive reactions, of which there were two, had good immediate and final results after eight months observation.

The six psychoneuroses all had good results, being relieved of their phobias or compulsions. The case of conversion hysteria did not do well.

For the whole group of 40 lobotomized patients, 37 improved immediately following lobotomy; three did not. After an average observational period of 28 months, 35 were improved and five unimproved.

Summary

(1) A series of schizophrenics who failed to respond to psychotherapy, chemotherapy or ordinary electroshock and/or insulin ther-

apy were given regressive EST. In the early years this was done inadvertently, but in recent years, deliberately. It was found that when patients were regressed the end results were better.

(2) Those cases which did not regress had a poor prognosis.

(3) It has been determined that there are various phases through which the patient *must go* in order to obtain relief from his psychosis. First, the remission phase, then the anxiety phase, followed by the regression phase. In a few cases it has been observed that a reanimation phase occurs. It is presumed that this is the final phase in the elimination of the psychotic picture but it takes persistence to carry the patient through weeks of the regression phase to await the reanimation phase. The subsequent phases of rehabilitation include those in the hospital, then in the home and the community and finally in the economic situation. During this period psychotherapy is administered.

(4) Patients who have a prolonged history of alcoholism or previous head injury should not be considered for regressive EST as they are poor risks.

(5) Forty patients who were refractory to long, heavy or repeated courses of EST and/or insulin therapy were lobotomized by the Grantham technic. A significant number improved as contrasted to those who obtained poor results.

(6) The lobotomized cases which did best were the paranoid, the catatonic and schizoaffective types of schizophrenia, the involutional psychoses, the depressive reactions, the psychoneurotic phobic reactions and the obsessive-compulsive neuroses. The manic-depressives did not do particularly well.

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Pleasure, Pain and Human Relations

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This paper seeks to define some of the interactions of human thoughts, feelings and deeds with the world in which we live. It attempts to do so partly by using the concept of social roles, the modes of behavior each society requires of its members. It proposes that the interactions between people resulting from their attempts to carry out these roles can be seen as based on varying combinations of two fundamental types of objective interpersonal relationships: mutual relationships and winner-loser relationships.

This paper also suggests that man's inhumanity to man—derived from the existence of objective winner-loser relationships in society—will continue so long as one human being is essentially an irrevocably and severely painful stimulus to another because his role requires him to be. This will be the case so long as the social structuring of roles requires one man to exploit another.

The inseparability of thinking, feeling and doing becomes ever more clear as time passes, but it was Sherrington,[1] who long ago pointed out that "every cognition has, potentially at least, an emotive value, and, along with that, conative effort as a further factor." Emotion is physiologically the motivating power of human thinking and doing. But what is this psychological entity, "emotion"? Can it be analyzed into simpler elements?

Pleasure and Pain: The Fundamental Feelings

The concept that pleasure and pain are the basic emotional elements is at least as old as Jeremy Bentham. But this concept has recently been reinforced by neurophysiological work[2] suggesting that there are two separate, although interdigitating, systems extending throughout the central nervous system which govern pleasure and pain respectively.

Lashley[3] has suggested that "the psychological data must be analyzed . . . with reference to the patterns of activity which they represent." What are the "patterns of activity" represented by pleasure and pain, the two fundamental elements of emotionality, the motivating force of psychology?

We propose that the "patterns of activity" underlying pleasure and pain are the basic motor patterns of approach and withdrawal, respectively.

The "pleasure" or "reward" system within the central nervous system can be seen, then, as having developed phylogenetically essentially, although not completely, in conjunction with refinement of the basic motor responses of approach. The "pain" or "punishment" system, *which has priority,* can be seen in contrast as having developed phylogenetically in conjunction with progressive refinement of the basic motor responses of avoidance. The pain or punishment system can be seen, then, as the primary initiator of emergency "flight or fight" reactions.

This hypothesis seems valid when the differences in response to painful and to pleasurable stimuli are compared, and when it is seen how similar these differences are to the differences between approach and avoidance responses. Dollard and Miller[4] have pointed out that when the intensity of response is plotted against distance from the stimulus, the slope of the avoidance curve is much steeper than that of the approach curve. From this we can perhaps understand why "avoidance" responses, presumably governed by the "pain" system, seem clinically to be more intense and urgent, although of briefer persistence, than the "approach" responses. The latter, presumably governed by the "pleasure" system, seem on the other hand to be gentler, although operative over greater distances and times.

Hence we can postulate that the basic emotional responses which drive our activities—pleasure and pain—are evolutionarily derived from the basic motor activities of approach and avoidance respectively, that each of these two different kinds of response is

governed by its own set of laws, and that any active response in real life involves the interaction of the two.

Interpersonal Pain and Pleasure

The first important interpersonal relationship of every individual in every society is both pain-removing and pleasurable. This first interpersonal experience for any of us is, of course, the experience of nursing. Long ago, Freud and others pointed out the hunger-pain-relieving quality of the nursing experience. But only recently has Harlow's[5] brilliant work separated the specifically pleasurable aspect of this experience—cuddling—from its pain-relieving aspect, relief of hunger-pain. From Harlow's work we can postulate that the *unconditioned* stimulus to the pleasure system is contact touch, or cuddling, and that the stimuli associated with nursing, beginning with those associated with the mother, become pleasurable *conditioned* stimuli because of their association with the *unconditioned* pleasure system stimulus, cuddling. Cuddling, then, is the unconditioned stimulus or experience upon which human love, or man's being a pleasurable stimulus to man, is founded. Needless to say, the stimuli associated with the nursing experience also acquire a pain-relieving quality as well.

What then causes man to be painful to man? What is responsible for the "alienation" of man from man, which the existentialists bemoan so loudly?

We are all born with the capacity to respond to painful stimuli, and we all, of course, experience them, beginning with hunger in the newborn. Painful experiences due to other people do not, however, usually occur until some time subsequent to the infant's having experienced interpersonal pleasure.

A painful quality of mother to child may begin in the feeding relationship, or it may not commence until toilet training is attempted, or it may not appear until awareness of sexual differences occurs. Although some interpersonal pain must occur in each person's life in order for him to learn natural and social reality, perhaps it is possible, with our present-day sociological and psychological knowledge, to keep this pain at a minimum, and to postpone, or even perhaps completely to avoid, the occurrence of severe, crippling interpersonal traumata.

Shortage as a Cause of Interpersonal Pain

One important reason for interpersonal pain is shortage. In times gone by, when food supplies were inadequate, the needs of one person could be fulfilled only if another person got less than he needed. Had the shortage not existed, it might have been objectively possible for each man to get what he needed (which must be distinguished from what he may have wanted) and no conflict, with resultant interpersonal pain, would have resulted.

Shortage, can, however, be artificially and socially created. The infant who is accustomed to being cuddled for six or seven hours a day may well experience a "shortage," which will be painful, if he suddenly is cuddled for only twenty minutes a day. Reduction of pleasures to which we have become accustomed can be painful in itself.

In adults, also, shortage is often artificially created. Basic biosocial needs can be frustrated by anti-human social laws or codes. The old social regulations decreeing the binding of the feet of upper-class Chinese women, the social regulations denying marital love to certain religious leaders, the social rules of conduct dictating expenditures beyond one's means in order to maintain one's status in a particular group, are all examples of the frustration of basic biosocial needs by anti-human social codes, whether they be explicitly defined or not.

The crippling of the feet of Chinese noblewomen tended to make them helpless and produced a "shortage" of their ability to take care of themselves and their children; consequently their need for a servant class beneath them was increased. The denial of marital love to certain priests results in an inevitable sexual "shortage"; if the sexual drives are satisfied illicitly, a burden of guilt is created, and if they are not, the torments continue, even though sometimes directed

into other channels. The social requirement to spend beyond one's means sets up "shortages" within the family economy, between the breadwinner and his source of income, and among the different members of the family, each of whom experiences an artificially created shortage.

It would appear, therefore, that shortage, naturally or socially created, may produce destructive human interaction, and may consequently be an important reason for man's inhumanity to man. It also appears that almost all shortage today is artificially created and maintained, inasmuch as man's conquest of nature has reached so high a level as to make natural shortage no longer necessary.

As we have seen, Lashley[3] suggested that "the psychological data must be analyzed . . . with reference to the patterns of activity which they represent." We have attempted to indicate the usefulness of defining this data in terms of the patterns of physiological motor activity, but we can also attempt to define it in terms of social activity as well.

How can interpersonal pleasure and pain, and the shortage which at least partly underlies interpersonal pain, be conceptualized in social terms?

Mutual and Winner-Loser Relationships

It appears useful to describe two fundamental elements or aspects of human relationships, both present, although in varying degrees, in almost every human relationship. One elemental aspect of a human relationship can be called its "mutual" aspect, which is comparable to the "I-Thou" relationship of Martin Buber. Here, the pleasure of accomplishment of the biosocial role of one individual is achieved *by virtue* of the other person's fulfillment of his biosocial role. Every society is based on the existence of such objectively mutual relationships, as between mother and child, teacher and pupil, husband and wife, doctor and patient, and worker and worker.

Out of relationships which are objectively mutual, and accurately and appropriately experienced as such, come feelings of interpersonal pleasure, warmth and even love, in its broadest sense. These are the *subjective* consequences within both partners of the accurately perceived *objective* mutuality of the relationship.

While such feelings of warmth may have a heterosexual element within them, they are in fact far more than mere sublimated sexuality. For this reason it would appear profoundly incorrect to disdain the warm feelings which develop between doctor and patient, for example, as "basically sexual," and therefore just a wee bit off-color.

The *winner-loser* aspect of human relationships involves one person's being able to fulfill his objective biosocial role *only at the expense* of another person's fulfilling his. Such objective conflicts between the *roles* of individuals are seen most frequently in overly competitive societies. One example of a relationship which is objectively winner-loser in character is that between two salesmen, each trying to sell the same limited territory. Here one man's success can be achieved only at the expense of the other's.

Even if the territory were adequate to support both salesmen, but one of them believed he had to have it all, and actively sought to achieve this end, a relationship which had the potentiality of being mutual would thereby be unnecessarily converted into a winner-loser one.

Winner-loser relationships can be compared to Buber's "I-it" relationships: the winner treats the loser like a thing. In them, as we see, the loser suffers pain while the winner obtains pleasure, the pleasure of achieving or obtaining that which he needs or wants. The winner fulfills his role, the loser does not. But the pleasure of the winner is not unalloyed; it is, rather, mixed with fear—anticipated pain—of being conquered in the future, as he himself has conquered in the past. In contrast to the conditional and unstable pleasure involved in winning in winner-loser relationships, the pleasure involved in truly mutual relationships lacks in itself any element of anticipatory fear.

Subjective and Objective Aspects Of Winner-Loser Relationships

In our society, although there are relatively few relationships which are in themselves winner-loser in nature, there are many relationships, in themselves objectively mutual, which are incorrectly *experienced* as winner-loser. After being incorrectly perceived, and reacted to, as winner-loser, they may then actually acquire an inappropriate winner-loser character. Winner-loser struggles between husband and wife, mother and child, doctor and patient, and teacher and pupil are examples of such situations.

How do relationships which are originally and objectively mutual in basic character deteriorate into subjectively and then objectively winner-loser relationships? Among the *social* causes are vague fear—ignorance, dogmatism and confusion of roles. The failure at times of psychoanalysts and other experts precisely and correctly to define the different biosocial roles in our society may perhaps sometimes have added to this confusion of roles. The *physiological* basis for this deterioration of mutuality into conflict is, it appears, the biological priority of painful stimuli over pleasurable ones.

The Priority of Painful Interpersonal Responses

The existence of two distinct neurophysiological systems within us, governing pain and pleasure respectively, has been pointed out, and the earliest unconditioned interpersonally-related stimuli to both systems have been mentioned: hunger-pain and cuddling-pleasure.

Each of us has experienced both pleasure and pain in his dealings with other people. Other people thus act as painful and/or pleasurable stimuli to a given individual.

But our neurophysiology is such that *in the presence of a mixed stimulus, we initially react excessively to its painful aspect.* In interpersonal terms, our instantaneous reaction to another human being exaggerates the painful stimuli he may be emitting, while under-evaluating the pleasurable stimuli he is also sending out at the same moment. Hence a person will tend to react *instantaneously* even to a friend to some extent as though he were a threat. Overt, immediate response to one's initial, instantaneous feelings will, therefore, tend to be excessively hostile, and to have a rather disruptive effect on interpersonal relationships.

Reflecting on the interpersonal situation, however, and the use of consciousness—verbalization, either to one's self or to others—makes it possible for more accurate appraisal of the pleasurable and painful aspects of the other person to take place. This more accurate, later appraisal occurs all the time, and serves to correct the *consistent distortion present in the initial response,* in which the painful quality of the stimulus person is thus exaggerated. This is why it is advisable to think before one speaks—even though the psychoanalytic focus on *initial* responses sometimes tends thereby to foster speaking *before* thinking. This focus also tends to place *an exaggerated importance on the initial feeling response,* without properly taking into account the inevitable distortion present within it.

The most primitive and most rapid responses of any organism to pain are the "fight or flight" reactions described by Cannon. The more fatigued, ill, tense, or panicked the organism is, the more easily are these primitive responses evoked. While these can take on rather sophisticated forms in the drawing room or analytic office, they are basically only attempts to escape from the individual being experienced as painful, or else to inflict greater hurt upon him than he has inflicted upon us. An impulsive, vindictive response to interpersonal pain is, however, by our definition, winner-loser in character. The spontaneous, initial response to interpersonal pain, therefore, is one of behaving as though the relationship were winner-loser in character.

But, as we have seen, our *initial* perception of the pain the stimulus person has inflicted is exaggerated by the nature of our physiology. If we fail to reckon with this distortion, and, acting upon our immediate feelings, seek to hurt the other person as much or more than we feel he has hurt us, a wholly

unnecessary conflict may result. This conflict may seriously interfere with, or even completely disrupt, useful and mutual communication between us.

Two Illustrations

Let us take some hypothetical examples to show how the initial overreaction to the painful, or hostile, aspects of a mixed interpersonal stimulus can impair, or even destroy, a relationship.

A new boy has just come into our Johnny's class at school. At the end of the day, he asks Johnny a question about the assignment for the next day, but seems to scowl as he does so. Asking the question, and having it answered, are two parts of a mutual relationship, in which one student helps another, thereby enhancing his own role as a good citizen without pain to himself.

Since interpersonal pain has priority at the moment of interaction, however, Johnny's first impulse may well be to scowl back at the new boy, but a little harder. If this occurs, and it makes the new boy somewhat angry, he may in turn respond in the same vein. An objectively mutual situation has then begun to become a conflictual one; despite the absence of objective role conflict between the two boys, the relationship between them has begun to assume a winner-loser character.

Of course, there are specific factors which will affect the likelihood of Johnny's scowling back rather than behaving in a more friendly way. The more fatigued, tense or frightened he is, the greater will be the chance that he will respond in a blind, disruptive fashion. The more he has been brought up "freely to express" his impulsive *initial* responses, the greater will the likelihood be that disruption of the relationship will occur in this way. The calmer and more knowledgeable Johnny is, however, the greater will be the chance that the initial, disruptive scowl will *not* occur, and that the relationship between the boys will proceed along the path of mutuality.

The same choice of paths for a relationships can occur between adults as well, although here the phenomenon is more subtle and complicated. Let us say that a rather arrogant new person in a social group knows more about something than I do. What responses are open to me?

My most spontaneous response is annoyance at his arrogance—his unspoken way of seeing us in competition, with himself as winner over me. In our competitive society, it is also easy for me to be irritated at him for knowing more than I, even though in a drawing room his greater knowledge need not make the relationship between us winner-loser in character. If for one or both of these reasons I allow my irritation to guide my response, my tendency may well be gently—or not so gently—to belittle, ridicule or otherwise attempt to humiliate him, inasmuch as feeling irritated by him, I feel myself in conflict toward him. The resultant interaction can be described as "one-upmanship," with each of us vieing to see who humbles the other more.

There is, however, another, more conscious and deliberate response available which is far more worthwhile. Despite my justified irritation at his arrogance, and despite any unjustified irritation because he knows more than I, it is possible for me to restrain my immediate, hostile response. Then, after recognizing the absence of any *objective* threatening elements in the relationship, I can respond in more friendly fashion. Even though his tone may be rather irritating, it is possible for me to recognize that he is no threat to me. Since this is the case, it is possible that the relationship can be led along mutual, rather than winner-loser lines.

One way to do this might be for me, in a dignified and forthright fashion, and without belittling myself, to take a pupil's role with him, and to learn from him. This would enhance his role as a man of knowledge by enhancing my own role as a searcher after it. Thus can a soft answer turn away not only wrath but also arrogance. But for such mutuality to be established under these rather difficult conditions, I must carefully refrain from responding to my initial, overhostile first impulses; instead I must evaluate the objective nature of the relationship

between us, differentiating between subjectively painful stimuli—such as his arrogant air—and the objective nature of the relationship itself.

Of course, there may be a real situation when the new man's superior knowledge *is* an objective threat to me; in this case, my response will of necessity be quite different. Such a situation will usually be the result of shortage, however. If, for example, he and I are both seeking the same position, and the one who makes the better impression on the potential employer will get it, my role will require that I do my best to show my superiority over him in this objectively-determined winner-loser relationship. Of course, the way in which I handle myself will depend in large measure on how much I stand to lose if I do not get the job; my tactics will tend to be far more ruthless if obtaining this particular job is necessary to avert starvation than they would be when many jobs are available.

The attitudes and behavior of another person can act as painful signals to any one of us. Our initial reaction will exaggerate the amount of pain in the situation; if we respond immediately without recognizing this built-in distortion, we are liable to react in a very erroneous way. If we thus overact to the painful aspect of interpersonal situations, while neglecting their pleasurable and mutual aspects, hostility and destructive competition can develop quite unnecessarily.

Reflection can therefore prevent disruptive overreaction to another person's annoying traits by helping us recognize the absence of *objective* conflict between us. This reflection will, of course, involve evaluation of the roles involved, and differentiating the painful quality of the stimulus person from his objectively unthreatening role. This we do in our analytic offices every day: when a patient is angry, and we find that there is no basis for this anger in terms of objective role-conflict between us, we softly present to him the idea that his painful, angry tone is an indication of overreaction on his part. Soft answers turn away wrath; spontaneous answers often tend to aggravate it.

Mutuality and Equality

Throughout this paper mutuality has been presented as socially desirable. Mutual roles have been defined as those in which the accomplishment of one person's biosocial role is achieved by virtue of the accomplishment by the other person of his biosocial role, and the mother-child relationship has been cited as an example. Here, quite obviously, the relationship is a most unequal one, in terms of the unequal capacities of the two people involved to determine the activities of the dyad. While the infant's biological needs, and the mother's biosocial needs, also, establish the foundation of this relationship, it is shaped far more by the mother—who will determine, for example, whether to feed with breast or bottle—than by the infant.

Similarly, a mutual relationship between adults need not be an equal one, and indeed usually is not. It is a major source of error to equate mutuality with equality, as, for example, in the relationship between doctor and patient in psychiatry. Although this relationship is indeed a mutual one, by no stretch of the imagination can it be considered an equal one. The fact that the patient's status and power are unequal to the doctor's are shown by the fact that the patient comes to the doctor's office, pays him, and behaves along the lines the doctor indicates. When a person is extremely incapacitated, the doctor's role in the relationship is proportionately more significant, and the inequality is greater. As the patient improves, however, he becomes more capable of making his own decisions, and the doctor's concepts become less "orders" and more "advice," even if the doctor does not directly "give advice." At the successful termination of psychotherapy, the patient has reached his closest to the doctor's level, but the relationship between them even at this time is not an equal one. If the couch is used, the inequality in status is even more strongly emphasized and thereby augmented.

It has been suggested elsewhere[6] that the equal value of people—before the law for example—must be distinguished from the unequal value, or power, of their roles. "While

in a democracy all *men* are created equal at the polls or before the bar," it was pointed out, "in no organized society are the *roles* of president and unskilled laborer equal in social importance. While the president and the laborer each have the same rights as *people* before the law, the *role* of the former is by far the more important."

Hence in almost all relationships which are mutual, there will still be inequalities of role within them; in the doctor-patient reationship, even if the President of the United States is the patient, his role is still that of the patient, and is therefore subordinate, within this relationship, to that of the doctor.

Mutuality is, therefore, based on *correct* perception of unconflicting social roles and their interaction, and these roles will usually *not* be equal in any particular one-to-one relationship, despite the fact that the roles of voters at the polls *are* equal in democratic societies.

Summary

From an examination of the physiology and sociology of human interactions, it has been postulated that viewing human relationships as combinations of winner-loser and mutual relationships is apparently of value. Mutual relationships are pleasurable to both parties, whereas losers in winner-loser relationships are pained by the other individual in the relationship. The winner in a winner-loser relationship gets pleasure, but this pleasure is alloyed by the fear of being conquered in the future as he has conquered in the present.

Because of the priority of the physiological response to painful stimuli, initial responses to interpersonal situations tend to exaggerate the painful aspect of the stimulus person. Acting immediately on the basis of this distorted response tends unnecessarily to disrupt relationships.

Conscious evaluation of painful stimuli through determination of the presence or absence of objective role conflict can help prevent such annoyances from interfering with warmth and mutuality.

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Evolution of the Care of the Mentally Ill

PERCY G. HAMLIN, M.D.

"I have here only made a nosegay of culled flowers and have brought nothing of my own but the thread that ties them together." Montaigne: Of Physiognomy, Chapter XII.

Manifestly a comprehensive account of this subject within the compass of a brief paper is not possible. A vignette only will be offered in the hope that the reader will be tempted to look longer and further to satisfy his intellectual appetite.

From earliest times mental disorder has been regarded by the multitude as a thing apart from bodily disorders, chiefly, no doubt because of its intangibility. It is indeed possible to say that the real civilization attained by a community can be judged by its attitude towards mental disorder. In order to produce data proving this it is necessary only to move through a few degrees of latitude or longitude to find a community small or large, at a differing stage of evolution, which holds the same views of mental disorder today as those current in Europe in the Middle Ages and before.[1]

Whitwell[1] (1936) says that in Shoa (Ethiopia) insanity, epilepsy, delirium and hysteria are always traced to demoniacal possession. In Mongolia cases of homicidal mania are regarded as persons possessed by "Damchan." In Syria mental disorders are treated by priests by means of exorcism and branding. In Malaya the bezoar stone, so prominent in medieval medicine in Europe for the treatment of mental and bodily disease is still held in high value. In Egypt the medicinal virtues of "mummy," considered to be of great value in Europe in the fifteenth and sixteenth centuries, are still accepted by many. In Polish Pomerania, succubi and incubi, under the name "Mora" are rampant just as they were all over Europe in the Middle Ages. In the Landes district of France the peasants still hang crosses of St. John's wort* over the door to keep evil spirits away. In Celebes, epilepsy is regarded as due to the soul leaving the body and in parts of Ireland it is still thought due to demonical possession.

And so it is all over the world according to the degree of real civilization attained. These examples merely show the various stages of evolution through which cultures have to pass in the laborious climb from magic and its brotherhood to science.

There frequently appear, like coral reefs, in the full tide of civilization little atolls of aberrant thought, not based upon ignorance, superstition or mental inertia, as in medieval days, but rather upon a peculiar type of mind, a kind of mental "sport" having a religious trend, a tendency towards fanaticism and an unbalanced view of things. Such a mind is often given to unusual flights of fancy which frequently cannot be satisfied with cold, formal, prosaic logic, but seeks explanation and finds satisfaction in the unknown, surrounded by a haze of words not easily understandable by the average mind. These mental sports are not infrequently of the paranoid or schizoid psychopathic personality makeup. Examples of their productions are unusual cults, freak forms of religion, spiritualism, and the astrology and palmistry of our own time.

It is believed that the earliest reference (circa 1457 B.C.) to madness in the Bible is probably that in which it is threatened amongst other things as a punishment for those who should violate God's commands, "He shall smite thee with madness and blindness and astonishment of heart."[1] (Bible: Deut. 28:28.)

Saul's madness has been historically diagnosed manic-depressive psychosis. It terminated in suicide. David feigned insanity (carrying the implication that the Israelites were familiar with it), to escape from Achish, King of Gath. He simulated simple dementia, "scrabled on the doors of the gate

*Saint John's wort, flowering tops of *Hypericum perforatum*, used as a remedy for chronic catarrh and intermittent fever and as a discutient. Dunglison, Dictionary of Medical Science, Lea Brothers & Co., Philadelphia, 1893.

and let his spittle fall down upon his beard."

In the ancient world there is ample evidence that the Egyptians, the Greeks, the Hebrews and the Romans were well acquainted with the symptoms of mental illness. As to its causes and cure, there is little but superstition, ignorance and cruelty.

Aristotle was alive to the influence of fever in causing hallucinations or illusions giving an instance of the seeming appearance of animals on the wall of a house. If, according to Aristotle the patient is not very ill he recognizes the nature of the illusion, otherwise he is deceived. Aristotle compares this deception of the senses to what happens when the timid man under the influence of fear fancies he sees enemies from the slightest resemblance. Or when the lover under the influence of his affection sees the object of his love. In short, in direct proportion to the intensity of the passion by which a man is influenced is the resemblance by which he is deceived.

Hippocrates, among the ancients, seems alone to hold aloft the torch of scientific, that is, clinical discernment. He understood epilepsy in a clinical sense and to use our modern term "debunked" it as the "sacred disease."

"Men regard its nature and causes as divine from ignorance and wonder because it is not at all like other diseases. I see men become mad and demented from no manifest cause and at the same time doing many things out of place. They who first referred this disease to the Gods appear to me to have been such persons as the conjurers, the purificators, mountebanks and charlatans. Such persons using the divinity as a pretext and screen for their own inability to afford any assistance have given out that the disease is sacred. The disease is formed from those things which enter and go out of the body, and it is not more difficult to understand and cure than the others. Neither is it more divine than other diseases."

From this brief consideration of the ancient period we move to a scrutiny of the period of the Middle Ages. Generally this may be regarded as a vaguely defined period between the Ninth or Tenth century and the Sixteenth century. It was a period of stagnation or even retrogression in science generally and in medicine and psychiatry in particular. During this period the medical world was almost entirely under the sway of Humoral Pathology, Demonology, Astrology, Witchcraft and Sorcery.

Nowhere during the medieval period did the mentally ill find more understanding and better treatment than at the hands of the "heathen Moslems." In Europe the spirit of inquiry which had motivated the ancient Greek physicians, philosophers and scientists was almost dead. The medical man for the most part had surrendered his craft to the theologian and witch doctor. But in the Mohammedan East the torch of medical science lit by Hippocrates was still held aloft overspreading the darkness of superstition in the treatment of mental and physical diseases.

While the insane in Europe were being sent to churches and monasteries to be exorcised of their possessing demons, the Arab physician Avicenna was insisting that they were simply suffering from mental maladies and should be treated as sick persons. Avicenna came from the country near Bukhara (now in the USSR about 200 miles north of the Afghanistan border), then the capital, intellectual and cultural center of Persia (Samanid realm). He lived 980-1037 A.D. and wrote about one hundred works. In the greatest of these, "The Cannon, a System of Medicine," he gave a classification of psychiatric clinical states, containing thirteen separately described conditions, including epilepsy. His philosophy is based on Aristotle but includes neoplatonic ideas. Not only may the Moslem world claim this shining exemplar but followers of Mohammed, with what appears to be historical justice, may claim the establishment of the first hospitals for the mentally ill.

(The claim that such an institution was built at Jerusalem in the Fifth Century is without substantial foundation in history. The legend that a morostrophium or house for lunatics existed at Constantinople as early as the Fourth Century is likewise unverifiable.[2])

Deutsch[2] cites the instance of Benjamin of Tudela (Spain), a traveller of the 12th

century. This man gave an interesting account of an asylum for the insane that he saw in the city of Bagdad. Here poor patients were maintained and treated at the expense of the Caliphate.

The first European asylum exclusively for the care of the mentally ill of which we have precise record was built at Valencia, Spain, in 1408. This was accomplished by Fray Gope Gilaberto. According to D. Hack Tuke[3] it is probable that here the Spaniards were following the lead of the Mohammedans who founded and endowed similar establishments shortly after the proclamation of Islam. It is to be mentioned, however, that although the hospital at Valencia was the first in Europe exclusively for the mentally ill of which we have record, we do have definite record of six male mental patients confined in Bethlehem Hospital, England, as early as 1403.[3]

The modern period, our period in psychiatry, may be said to date from the French Revolution. It is true that Benjamin Rush usually spoken of as the Father of American Psychiatry, had made beginnings at the Pennsylvania Hospital in Philadelphia, just before the French Revolution. The humanitarian attitude towards the insane crystallized in a world-wide sense after Pinel and the French Revolution. The Salpetriere, the Bicerte and Charenton hospitals emerged in the Revolution and flowered into teaching institutions for students from all of the world. Teachers like Philippe Pinel, his son Scipio, Esquirol, Ferrus, Pariset and Georget led the movement. These French doctors contributed much towards making psychiatry a recognized medical discipline. Two generations later the fame of the neurologist Charcot attracted to the grimy old walls of the Salpetriere, a young Viennese doctor named Sigmund Freud.

Esquirol died in 1840. Four years later thirteen American mental hospital superintendents met at Jones Hotel, Philadelphia (Oct. 16, 1944), and formed the Association of Medical Superintendents for the Insane. They are known to history as "the Original Thirteen." In 1893 the title of the Association was changed to the American Medico-Psychological Association. In 1921 it was changed to its present name, The American Psychiatric Association.

The composition of the Original Thirteen was as follows: Samuel B. Woodward, Worcester State Hospital, Massachusetts; Isaac Ray, Augusta State Hospital, Maine; Luther V. Bell, McLean Asylum, Massachusetts; Charles E. Stedman, Boston Lunatic Asylum, Massachusetts; Neremiah Cutter, Pepperell Private Asylum, Massachusetts; John S. Butler, Hartford Retreat, Connecticut; Amariah Brigham, New York State Lunatic Asylum, Utica, New York; Samuel White, Hudson Lunatic Asylum (private), Hudson, New York; Pliny Earle, Bloomingdale Asylum, New York; Thomas S. Kirkbride, Pennsylvania Hospital for the Insane, Philadelphia; William M. Awl, Ohio Lunatic Asylum, Columbus; Francis T. Stribling, Western Lunatic Asylum, Staunton, Virginia; and John Minson Galt, Eastern Lunatic Asylum, Williamsburg, Virginia.

Woodward was the first president of the Association, White vice-president, and Kirkbride secretary. The Thirteen had a silent but amazingly helpful partner in that phenomenal and devoted woman, Dorothea Lyne Dix.

In studying the official reports of Dr. John M. Galt of Williamsburg to the Virginia Legislature through the years 1842-1850, the writer found that Dr. Galt knew and employed occupational therapy, recreational therapy, biblio-therapy and music therapy. He speaks of these and of books and games as "revulsives of utility in turning away the mind preoccupied by phantasy."

Later on our own American scene following the turn of the century were to come men like Adolf Meyer, MacFie Campbell, E. E. Southard, Thomas W. Salmon, Frankwood Williams, Clifford Beers, Wm. A. White, the Menningers. There came into being teaching institutions like the Phipps Psychiatric Clinic in Baltimore, the Boston Psychopathic, Psychiatric Institute, N. Y., St. Elizabeth's, Washington, D.C., the Menninger Clinic, Topeka, Kansas.

The evolution of the care of the mentally ill from the days of the Egyptians and Greeks to our own time has been one of institutions

and techniques, yes. But most of all it has been a revolution in the minds of men like Pinel, Rush, Clifford Beers and Adolf Meyer. Like Abou Ben Adhem, their name is to be written among those who love their fellow men.

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Fluphenazine Use with Chronic Psychotic Patients

JOSEPH A. BARSA, M.D., and JOHN C. SAUNDERS, M.D.

Tranquilizing drugs have two chief clinical actions: a sedative or calming action and an anti-psychotic (i.e., anti-delusional and anti-hallucinatory) action. Different drugs possess these attributes in varying degrees. The phenothiazine derivatives, which comprise the largest number of tranquilizers, can be divided into three groups according to chemical structure and clinical action. There is the di-methyl group (with two methyl radicals on the terminal nitrogen of the side chain), the piperazine group (with a piperazine ring in the side chain), and the piperidine group (with a piperidine ring in the side chain). The di-methyl group includes chlorpromazine (Thorazine), promazine (Sparine) and trifluprомazine (Vesprin); the piperazine group: prochlorperazine (Compazine), thiopropazate (Dartal), trifluoperazine (Stelazine), perphenazine (Trilafon) and fluphenazine (Prolixin); and the piperidine group: mepazine (Pacatal) and thioridazine (Mellaril). Listing the groups according to the strength of their sedative action, we have the di-methyl group, the piperidine group, and the piperazine group. However, in the order of their anti-psychotic effectiveness, they are: the piperazine group, the di-methyl group, and the piperidine group. The piperazine group has the greatest incidence of extrapyramidal side effects, especially akathisia; next comes the di-methyl group with predominantly parkinsonian symptoms, and finally, the piperidine group.

The present study concerns the use of fluphenazine in the treatment of chronic psychotic patients. As noted, fluphenazine is in the piperazine group of phenothiazine derivatives. From our experiences with other members of this group, we expected that the drug would have a strong antipsychotic effect and a relatively weak sedative effect. We chose, therefore, patients who were idle, seclusive, withdrawn, preoccupied, disinterested, apathetic, delusional and hallucinated. There were 57 female patients between the ages of 23 and 74. They had been continuously hospitalized for 3 to 28 years. Their diagnoses were as follows: 49 schizophrenia, 2 involutional psychosis, paranoid type, 2 psychosis with epilepsy, deterioration, 2 psychosis with cerebral arteriosclerosis, 1 psychosis with mental deficiency, and 1 alcoholic psychosis, Korsakoff's syndrome. All of the patients had been receiving tranquilizing drugs for at least two years with slight or no improvement. Twelve patients were receiving chlorpromazine, 14 a combination of chlorpromazine and trifluoperazine, 11 trifluoperazine alone, 7 prochlorperazine, 3 a combination of chlorpromazine and prochlorperazine, 4 perphenazine, 3 trifluprомazine, 1 thioridazine, and 2 reserpine.

At the beginning of the study all previous medication was discontinued. The patients were then placed on fluphenazine. This drug was given once a day in the morning at a starting dose of 1 mg. or 2.5 mg. or 5 mg. de-

From Rockland State Hospital, Orangeburg, New York.

The drug fluphenazine was supplied through the courtesy of E. R. Squibb & Sons as Prolixin.

pending on the amount of their previous medication. Thereafter, the dose was raised or lowered in accordance with each patient's response to the drug. The highest dose used was 10 mg. once a day. The study was continued for seven months. However, in the case of 28 patients fluphenazine had to be discontinued earlier because they became increasingly disturbed, restless, tense, irritable, hostile, and even aggressive. In 6 patients fluphenazine was stopped after one month, in 9 after 3 months, in 8 after 4 months, in 4 after five months and in 1 after six months.

The 29 patients who remained on fluphenazine were evaluated at the end of seven months as follows: 5 patients were markedly improved, i.e., in remission free of delusions and hallucinations and either released or ready for release from the hospital; 9 patients were moderately improved, i.e., although still delusional and hallucinated, they were now more alert, more interested in their environment, more sociable, and more active in the hospital program; 5 patients were slightly improved, and 10 patients were unimproved.

Side effects were similar to those previously encountered with other members of the piperazine group of phenothiazine derivatives, except that in the case of fluphenazine the side effects were more frequent and more severe. Fifteen patients experienced generalized tremulousness often associated with restlessness and agitation, usually early in therapy. One patient had what appeared to be severe shaking chills in the first 10 days. Another patient had myoclonic jerks involving the trunk and upper extremities. A third patient complained of severe dizziness during the first week. Still another patient developed marked rigidity, an unblinking stare and a protruding tongue. All of the above symptoms, with an exception mentioned below, responded fairly well either to benztropine methanesulfonate (Cogentin) alone or to reduced dosage of fluphenazine in addition to benztropine.

However, there was one side effect (if, indeed, it is a side effect and not one of the primary actions of the drug), which responded poorly to benztropine alone or even to reduction of dosage plus benztropine. We are referring to the peculiar stimulating action common to the phenothiazine derivatives in the piperazine group. It is this action which may be responsible for awakening the seclusive, withdrawn, apathetic, preoccupied patient and making the patient more alert, more sociable, more interested in the environment. Nevertheless, this action may produce (in some patients earlier than others) an increased restlessness, tension, irritability, hostility and aggressiveness; and in a number of patients there is also a generalized tremulousness. The tremulousness responds better to benztropine than do the other symptoms of this stimulating action. It was found necessary to discontinue fluphenazine in the 28 patients because of inability to control these symptoms. Furthermore, from our experience with fluphenazine and other drugs in the same group, we have found that the greater the anti-psychotic effect of the drug the more frequent and more severe is the stimulating side effect.

Blood and urine examinations revealed no abnormalities during the course of this study.

Conclusions

1. Fluphenazine is the most potent of the piperazine group of phenothiazine derivatives, and as such has strong anti-psychotic (i.e. anti-delusional and anti-hallucinatory) action and relatively weak sedative action. It has the added advantage of being effective in a once-a-day dose. It is most useful in those withdrawn, regressed schizophrenics who require potent anti-psychotic action without excessive sedation.

2. A high percentage of fluphenazine-treated patients (in our series 28 out of 57) develop a side effect that appears to be excessive stimulation, characterized by restlessness, tension, irritability, hostility and aggressiveness. This side effect is difficult to control and often requires discontinuation of therapy.

Imipramine in Office Management of Depressive Reactions

(A Double Blind Clinical Study)

E. ADAMS DANEMAN, M.D.

In evaluating the effectiveness of a method of treatment, that method must be compared to some standard. In evaluating the treatment of a psychiatric disorder, comparison with a standard treatment is especially important, and the comparison should be made under carefully controlled conditions to assure unbiased results.

It is to be regretted that few properly controlled studies have been reported in the literature on the increasing number of medications used in treating psychiatric states. Studies have been confined to hospitalized patients although it is equally necessary to appraise medications under office conditions, where results may be quite different from those obtained in a hospital. To cite an example, the resin-like substances used to remove sodium in the management of edema worked well in hospitals but failed in office practice.

While it is ultimately necessary to compare similar psychiatric drugs against each other, and against other recognized methods of treatment, the first step in evaluating a medication must be a controlled study of treatment *with* and *without* the drug in question. This is best done by administering either the drug or a placebo of similar appearance, in a double blind trial so that neither the patient nor the physician administering the drug knows which preparation is being given. Since April 1959 we have been making such an evaluation of imipramine (Tofranil—Geigy) in the office treatment of depressive reactions.

This drug bears a superficial chemical resemblance to the phenothiazin tranquilizers but differs in its central ring by the substitution of a two-carbon chain for the sulfur atom of phenothiazine. This substitution results in a remarkable change of pharmacologic properties. Imipramine has no tranquilizing effect, and indeed has little if any action on the central nervous system of normal individuals. It has a mild parasympathomimetic action that is responsible for most of its side effects. It is distinct from other antidepressant drugs in use in that it has no inhibitory effect on the enzyme monoamine oxidase.

Kuhn[1] in Switzerland first reported in 1957 that the drug had a specific beneficial effect on various depressive ractions. His original observations have since been confirmed by numerous clinical reports of other investigators, most of whom have stressed its effect in psychotic depressive reactions of a so-called "endogenous" type, in which it is reported to be successful in about 80 per cent of patients. Several reports mention that neurotic depressive reactions also respond to the drug.[2-5] To my knowledge, there has been only one controlled study of imipramine in out-patients, that of Ball and Kiloh[6] in England, published in 1959. Comparing imipramine and an inert placebo in a double blind study, they found a highly significant difference between the two compounds in endogenous depressions, and a substantial, though less significant, difference in neurotic ("reactive") depressions. The diminished significance in the latter case was doubtless due to the small number of patients studied, less than 25 on each compound.

Material and Methods

Patients in the present study were all seen and treated in office psychiatric practice on referral from other physicians. Patients were included in the study whenever a diagnosis of depressive reaction was made and no effort was made to exclude any patient with depressive symptoms. A diagnosis of neurotic depressive reaction was made in 159 cases, and psychotic depressive reac-

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Imipramine Hydrochloride (Tofranil) and matching placebos were generously supplied by Geigy Pharmaceuticals.

tion in 32 cases. Four additional patients were included with depression as the major factor associated with chronic brain syndrome, since it is our belief that depressive reactions in chronic brain syndrome represent psychological reactions to the organic defect.

The treatments specifically compared in the study were imipramine hydrochloride, in tablets of 25 mg., and placebo tablets of identical appearance, containing atropine sulfate 0.175 mg. Ball and Kiloh reported that with inert placebos it was often possible to identify the active compound by its pattern of side effects. To avoid unintentional identification of the drug in this manner, placebo tablets were compounded with atropine to mimic the side effects of imipramine. In prescribing the medication, care was taken not to give any special instructions that might encourage patients to expect any specific result other than certain atropine-like side effects, about which all patients were cautioned routinely. We felt obligated to instruct patients to anticipate these side effects, since previous experience had shown that many patients discontinue treatment if they are not forewarned. The dosage usually prescribed was four tablets daily (100 mg. imipramine or 0.7 mg. atropine), increased by one tablet each day until a dosage of eight tablets daily was reached (200 mg. imipramine or 1.4 mg. atropine). A low maintenance dose of four tablets or less was often achieved after a therapeutic response had been obtained. The patients were treated with one or the other preparations according to a random process of selection. At the outset imipramine and placebo for the study were stockpiled at a local pharmacy, and the pharmacist was provided with a deck of shuffled cards, half designated imipramine, half placebo. Special prescriptions for imipramine were issued to the patients, to be dispensed with imipramine or placebo as instructed on the next card.

Only the pharmacist knew the final arrangement of the cards, so that neither the patient nor the physician who evaluated the response could know which treatment was being used. In this way we were able to meet the requirements of a double blind comparison, and the patients were arbitrarily divided into two treatment groups. These two groups were generally comparable with respect to the type of depression diagnosed (Table I), although there were slightly more neurotic depressions in the placebo group, and slightly more psychotic depressions in the imipramine group. The two groups were also comparable with respect to age (Range: 17-75 years) and sex (31 per cent male). Presenting symptoms were found on subsequent analysis to have been somewhat more severe in patients assigned to the placebo group than in those assigned to the imipramine group, but this did not affect the conclusions drawn from the study since the response to treatment did not differ significantly with the severity of symptoms.

TABLE I

Depressive States Treated

	Group A Imipramine		Group B Placebo	
	No.	%	No.	%
Diagnosis				
Neurotic depressive reaction	70	74.5	89	88.1
Psychotic depressive reaction	21	22.3	11	10.9
Depression with organic disease	3	3.2	1	1.0
Total	94	100.0	101	100.0

In addition to the particular treatment under study, each patient continued to receive all other supportive and therapeutic procedures customarily employed and interviews with relatives were continued. In particular, we have given most of the patients in this study one of the phenothiazine tranquilizers, and chloral hydrate was often used to promote sleep at the beginning of treatment.

Fundamental to our practice is the use of psychoanalytically oriented psychotherapy, aimed at understanding the patient's hostility as directed toward the incorporated object-image. We have therefore continued our procedure of seeing each patient once or twice a week in interviews lasting 45 minutes. Evaluation of each patient's prog-

ress was made at weekly intervals during the first month of treatment, and at somewhat longer intervals thereafter.

Evaluations were recorded on the Malamud-Sands rating scale and also, for purposes of this study, on a specially devised scale for rating depressive symptoms (Figure 1). This scale was suitable for evaluating the change in severity of eleven specific symptoms on a 4-point scale, to form the basis of an over-all evaluation as excellent, good, fair, or poor. The term *excellent* was reserved for essentially complete remission of symptoms, *good* and *fair* for partial remission, and *poor* for no change or aggravation of symptoms.

Antidepressant Evaluation

Symptoms:

Sadness (or mood depression)
Psychomotor inhibition or "Slowing down"
Loss of interest in life
Self-reproaches
Crying
Pain
Sleeplessness, insomnia
Worry & diffuse concern
Fatigue, lack of energy
Appetite loss
Agitation, anxiety

Side Effects:

Drowsiness
Dry mouth
Other

Excellent ☐ Good ☐ Fair ☐ Poor ☐

Opinion:

Figure 1.

Patients were included in the final tabulation of results only if there had been at least three follow-up evaluations with the rating scale. Few patients were lost to follow-up and the only other patients not included in the tabulations were individuals not assigned to either treatment group because they failed to have prescriptions filled.

Results

The relative effectiveness of the two types of treatment is indicated by comparing the dosages of imipramine and placebo on which patients were maintained (Table II). It is apparent that dosage could seldom be reduced below 6 or 8 tablets daily in placebo patients, while more than 40 per cent of the imipramine patients could be maintained on 4, 3, or even 2 tablets. There were 12 patients in the study who did not receive medication long enough to arrive at a maintenance dose. The duration of treatment in the two groups also reflects to some extent the effectiveness of the two treatments. About one third of the imipramine patients were still receiving the drug after 90 days of study, while treatment with placebo had been abandoned in all but nine patients.

TABLE II

Dosage and Duration of Treatment

Maintenance Dose (No. tablets per day)	Group A Imipramine No.	%	Group B Placebo No.	%
2	9	9.6	—	—
3	3	3.2	—	—
4	27	28.7	10	9.9
6	23	24.5	22	21.8
8	22	23.4	67	66.3
Not maintained	10	10.6	2	2.0
Duration of Treatment (Days)				
Up to 30	34	36.2	17	16.8
31 - 60	16	17.0	49	48.5
61 - 90	12	12.8	26	25.7
Over 90	32	34.0	9	8.9

The over-all response to the two drugs was determined at the end of one month, and again at the end of two months of treatment, in all patients who were seen at those interlals (Table III). In this way the effects of imipramine and placebo could be compared directly. After one month of therapy, 79 per cent of the imipramine patients were in complete remission, as compared with 7 per cent of the placebo patients. There was only one failure in the imipramine group, while there were 39 failures (43 per cent) in the placebo group. These differences are of

TABLE III

Response to Treatment

After 1 Month	Total Evaluated	Excel. #	Excel. %	Good #	Good %	Fair #	Fair %	Poor #	Poor %
Imipramine	94	78	62	79.5	11	14.1	4	5.1	1	1,3
Placebo	101	90	6	6.7	10	11.1	35	38.9	39	43.3
After 2 Months										
Imipramine	94	54	47	87.0	6	11.1	1	1.9	0	—
Placebo	101	48	5	10.4	4	8.3	14	29.2	25	52.1

course highly significant statistically (P being less than 0.01). A two-month evaluation was possible in slightly more than half of the original number of patients, with an equally striking difference between imipramine and placebo (P being less than 0.01).

Notice that after two months there were no failures in patients taking imipramine.

Most of the patients studied were suffering from neurotic depressive reactions. When results were compared in these patients after one month of treatment, 78 per cent of 60 imipramine patients had obtained complete remission, only 6 per cent of 80 patients on placebo. After two months, remission was complete in 85 per cent of imipramine patients and 12 per cent of placebo patients. These differences are also statistically significant (P is less than 0.01). When a final evaluation was made at the end of treatment (Table IV), bearing in mind that treatment lasted for varying periods up to nearly a year, we see that approximately 80 per cent of the imipramine patients had attained complete remission of symptoms but only 4 per cent of the placebo patients.

It is of interest to examine the effect of treatment on specific symptoms associated with depression. Episodes of crying were controlled fairly well by placebo, and insomnia was controlled to some extent in the placebo patients by chloral hydrate. Both these symptoms were controlled much more effectively by imipramine, as were all other symptoms listed, including various painful symptoms. In each case the dfference between imipramine and placebo is highly significant statistically (P less than 0.01).

The side effects of imipramine and atropine placebo that were observed during our study (Table V) indicate that we chose a dose of atropine (0.17 mg. per tablet) almost precisely equivalent to the atropine-like activity of a 25 mg. tablet of imipramine. Nearly all patients experienced some undesirable effect of medication, usually dryness of the mouth. It is our impression that those patients with early onset of dry oral or pharyngeal mucous membranes also showed an early onset of improvement of depressive symptoms. Dry mouth was complained of less frequently on doses of 100 mg. or less, and also appeared to decrease with continued therapy.

A substantial proportion of patients experienced drowsiness, constipation, or blurred vision which effects were often not volunteered by the patient and were elicited only on questioning. A saline laxative in half dosage usually gave good results in constipated patients. It should be noted that patients had to be warned not to try to have refractive errors (blurred vision) corrected

TABLE IV

Final Result of Treatment

	Imipramine		Placebo	
	Number of Patients	Percentage Remission	Number of Patients	Percentage Remission
Overall Responses	94	79.8	101	4.0
Specific Symptom Responses				
Sadness	89	78.7	101	4.9
Psychomotor inhibition	91	78.0	100	7.0
Loss of interest in life	88	79.5	101	7.9
Self-reproaches	81	76.6	79	13.9
Crying	58	93.2	80	25.0
Pain	26	65.5	44	13.6
Insomnia	79	83.6	98	30.6
Worry, diffuse concern	91	71.4	100	6.0
Fatigue	92	76.2	100	3.0
Anorexia	72	86.1	99	14.1
Agitation, anxiety	80	82.5	95	10.5

TABLE V

Incidence of Side Effects

(PER CENT)

Effect	Imipramine (94 Patients)	Placebo (101 Patients)
Dry mouth	85.1	73.3
Drowsiness	30.9	43.6
Constipation	17.0	24.8
Blurred vision	9.6	17.8
Agitation	9.6	4.0
Hallucinations	1.1	—
Sweating	1.1	1.0
Photosensitivity	4.2	4.0
Dermatitis	2.1	—
Muscular weakness	1.1	—
Total incidence of side effects	91.5	80.2
Incidence of effects requiring cessation of treatment	4.3	—

during imipramine treatment. Several patients had gone to considerable expense to correct refractive errors.

Symptoms of anxiety or agitation occurred during treatment in nine patients on imipramine and four patients on placebo. The drug had to be discontinued in four imipramine patients because of the development of extra-pyramidal symptoms with consequent severe agitation; these four patients represented the only occasions when imipramine had to be withdrawn because of severe side reaction. Two of these patients were started on rather high doses (200 mg. daily) and one had obtained complete remission before the drug was withdrawn. Since phenothiazine tranquilizers were discontinued at the same time in all four cases, it cannot be stated categorically that the syndrome was caused by imipramine.

It is of interest that four patients on each treatment complained that they had become sunburned more easily during treatment. It was necessary only to advise these patients to keep their bodies well covered outdoors.

Discussion and Conclusions

In addition to carefully controlled studies of one medication, a dire need exists for comparative studies of various pharmaceutical preparations active in the same clinical area. Little work of this nature has been done; the present paper is the result of the evaluation of one drug, but it is anticipated that another study will begin at once, comparing this drug with others that may also be active against depressive reactions.

As suggested by Dr. Samuel P. Martin, of the College of Medicine at the University of Florida, it would be most interesting to compare imipramine and other new antidepressant drugs with amphetamines, combinations of amphetamines and barbiturates, and other standard medications for the treatment of depression. In addition, there is a need for a continuing evaluation and comparison of standard office procedures in use in various communities for the treatment of depressive states.

The study reported here justifies the following comments:

1. That imipramine is a safe and extremely useful drug in the office treatment of neurotic and psychotic depressive reactions, and has undoubtedly made hospitalization unnecessary in at least a third of our patients. In this respect it represents the best medication yet evaluated for the treatment of depression.

2. Side effects presented little problem in our study, since the possibility of atropine-like effects was explained in advance to each patient. It was necessary to discontinue imipramine in only 4 per cent of patients.

3. We believe that imipramine will find an established place in the office treatment of depressed patients, but wish to emphasize that it was never used without concurrent psychoanalytically oriented psychotherapy.

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The Six Herculean Labors of the Psychic Masochist

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Every school child knows that Hercules (Heracles) was an illustrious hero of Greek mythology who had to perform twelve labors and did so successfully. These tasks were the capture of the lion of Nemea, the killing of the nine-headed hydra of Lerna; the capture, alive, of the boar of Erymanthus; the capture of the hind of Arcadia; the routing of the man-eating birds of Mount Stymphalus; the capture of the girdle of Hippolyte, Queen of the Amazons; cleansing the stables of Augeas, King of Elis; the capture of the Cretan bull; capture of the man-eating mares of Diomedes; seizing the oxen of Geryones; obtaining the golden apples of the Hesperides; bringing the dog Cerberus up from the lower world.

All of the hero's troubles stemmed from the hatred of Hera, Zeus' wife and sister, who is represented in the mythology as jealous, vindictive and cruel. Hercules was the child of the extramarital union between Zeus and Alcmene, a mortal; as such he incurred Hera's hatred even before he was born. While he was still in his cradle he strangled two serpents she sent to kill him. In his teens he married Megara, but later killed her in "a fit of madness" induced by Hera. As punishment for this deed he became the slave and servant of Eyrystheus, King of Argos. The "twelve labors" were imposed on Hercules during his servitude.

The Hercules of mythology has a modern equivalent. That equivalent does not perform heroic deeds outside, but inside. The forum internum supplants the forum externum. Today's Hercules does not appear as a strong man but as an everyday and rather inconspicuous fellow. His feats are no less heroic for that, nor for the fact that they are visible only under the analytic microscope.

Who is homo masochisticus?

Externally, he can "pass" without difficulty. He dresses like everyone else, behaves like anyone else, has familiar likes and familiar dislikes. His intentions are of the best: he wants love, success, happiness. But—and this is one of the points at which the species branches off—something always (or nearly always) goes wrong. His ratio of success and failure shows a preponderance of failure, especially in decisive situations. At these times he *consciously* considers himself an innocent victim of outside malice. Peculiarly enough, he *unconsciously* seeks that malice out, uses and misuses it for his even more peculiar inner aim: failure, humiliation, defeat.

These really unbelievable people are addicted to an inner drama consisting of three acts.

First, unknowingly and unwittingly, by means of their own well-placed provocations (or by misusing a given situation), they will achieve a disappointment, a rebuff, a humiliating insult, a kick in the face.

Second, in blessed ignorance that they had unconsciously engineered the identical defeat that enrages them consciously, they will see only the other person's mean action and pretend that the whole cycle of events started with the enemy's "unwarranted aggression." Therefore they fight back, flushed with righteous indignation and certain that they are acting in self-defense.

Third, after the self-provoked defeat (the enemy is so chosen that psychic masochists cannot win) they will lament their wrongs and whimper in self-pity. The content of the complaint never varies: "Such an injustice! These things happen only to poor little me!" Again, these people are unaware that they have unconsciously achieved what they wanted: enjoyment of masochistic pleasure under the self-deluding guise of an absolving alibi.

The result of this trial is an endless chain of injustice collecting.

The pattern described above (which I have elaborated upon in several books and clinical studies, summarized in "Principles of Self-Damage," Philosophical Library, New York, 1959), corresponds to the *clinically* observ-

able behavior of psychic masochists. This "clinical picture" must be distinguished from the "genetic picture" explaining the historical development of the scourge; the latter was partly outlined by Freud, although he made little use of his own discovery.

As I see it, the trouble starts with two inborn, hence biological "tendencies": infantile megalomania and aggression. The infant and baby is endowed with more aggression than he can handle because of his undeveloped muscular apparatus. That aggression, triggered by any offense to infantile megalomania, which cannot flow outside turns against the child himself, and the problem is subsequently exacerbated by parental dicta and resultant guilt. That self-aggression is painful; later it is libidinized and thus made pleasurable. The picture is complicated by the fact that the inner conscience (superego) absorbs a good portion of the unusable total aggression of the child. Once in control of this store of aggression, it treats the child with the identical cruelty with which the child would attack the outer world if he had the power to do so. The ego meekly accepts this aggression, and once more aggression is converted into psychic masochism. This "genetic picture" is transformed into the "clinical picture" which rounds out the story of psychic masochism when the superego, in its anti-hedonistic aspect, becomes suspicious of the pleasure involved. As a result, the poor masochist must represent himself as the innocent victim of outside malice. The final complication in the procedure arises from inborn megalomania, which objects to self-humiliation. That objection is overcome by the fiction that the humiliator is merely the psychic masochist's executive organ (see Third Labor).

When the term, psychic masochism, is mentioned before either physicians or laymen who are not psychiatrically informed, knowing smiles tend to appear on the listeners' faces. These smiles of apparent recognition and understanding actually arise from semantic confusion: the uninitiated mistake psychic mesachism for perversion masochism and identify the "psychic masochist" as a man who pays a prostitute a double fee for whipping him. There are few perverted masochists; the aberration is not important enough to merit concentrated attention. Psychic masochism, on the other hand, is universal. There are as many psychic masochists as there are inhabitants of the earth (although, as George Orwell said, some are more equal—meaning here, more neurotic—than others). Psychic masochism does merit concentrated and prolonged attention. The psychic masochist does not want a physical beating, but emotional defeat, humiliation, failure. In contradistinction to the perverted masochist, he knows nothing of his noble aim, nor does he consciously enjoy the punishment he seeks. Quite the contrary.

The psychic masochist uses an enormous amount of cunning, cleverness and energy in pursuing his unproductive aim of being mentally kicked. Specifically, in the split second before inaugurating his masochistic fiesta he must perform no less than six labors.

FIRST LABOR: *Presenting oneself as innocent victim, hence maneuvering the other person into the position of refuser.* The psychic masochist's darkest secret is the fact that he himself is author, stage director and actor in the dramas of defeat which are his daily diet. He himself, therefore, must do the preparatory work of finding an opening for his provocation, or figuring out how to misuse a specific situation. He is a wizard at this job, and it is a job that requires mental work of high capacity.

To cite an example:

Under pressure from his wife, a 45-year-old corporation executive consulted me recently. His wife's complaint was that the man lived like a bachelor, coming home at unpredictable hours and refusing to discuss his wanderings, and that he treated their eight-year-old child coldly. She had made up her mind to change the existing situation or, after an "agonizing reappraisal," to divorce her husband.

The man told me that his snobbish wife was forcing him to live above his means. She had insisted on taking a luxurious apartment in a fashionable neighborhood and on sending their child to an expensive private school. Despite his excellent income, he was constantly in debt. Moreover, he hated his job and wanted to be a writer. He concocted this plan: His wife and daughter would spend the next

year or two living with his parents in Florida. (His parents lived on a modest pension; he admitted that their circumstances were very modest.) He himself would give up his job in New York and go to Hollywood, where he would try to establish himself as a television writer. What did I think of the plan? I told him that it had all the earmarks of middle-age revolt, and made sense only if he was prepared to divorce his wife. Why, I asked him, should she consent to the life of deprivation assigned to her in this scheme? I went on to suggest that he did not seriously expect his wife to agree to the idea, but actually wanted a refusal from her so that he could represent himself as the "innocent victim." Moreover, since he suffered from writer's block, wouldn't it be more logical to straighten himself out in treatment *before* giving up his job?

I told him of an analyzed case in which a writer, before entering treatment, had published an unsuccessful novel. Immediately after publication he suggested to his wife that he give up his well-paying job as college professor, retire with his wife and two children to a dilapidated shack in the country, and live on his "inheritance"—the interest, $100 a month, paid on a trust fund set up by his father. His wife quite reasonably answered that his plan would make sense after the publication of a successful, not an unsuccessful book. The violent conflict that followed brought the man into analysis. It could be established that inwardly he had never believed that his wife would be amenable to his grand plan. Unconsciously, he had wanted to push her into a position in which she would have to be "unjust" to him.

SECOND LABOR: *Display of pseudo-aggression.* The psychic masochist is very active in pursuing a passive goal, that of being kicked and humiliated. After having repressed his initial provocation, he attacks the provoked person with viciousness and determination, totally convinced that he is acting in self-defense for the protection of his inalienable rights. The aggression he displays, however, is false; it is not real but pseudo-aggression. This table illustrates the difference between the two types of aggression:

Normal Aggression	*Neurotic Aggression (Pseudo-Aggression)*
1. Used only in self-defense.	1. Used indiscriminately when an infantile pattern is repeated with an innocent bystander.
2. Object of aggression is a "real" enemy.	2. Object of aggression is a product of fantasy, or artificially created enemy.
3. No accompanying unconscious feeling of guilt.	3. Feeling of guilt is always present.
4. Dosis: amount of aggression discharged corresponds to provocation.	4. Dosis: slightest provocation, greatest aggression.
5. Aggression always used to harm enemy.	5. Pseudo-aggression often used to provoke "masochistic pleasure" expected from enemy's retaliation.
6. Timing: ability to wait until enemy is vulnerable.	6. Timing: inability to wait, since pseudo-aggression used as defense mechanism against inner reproach of psychic masochism.
7. Difficult to provoke.	7. Easily provoked.
8. Element of infantile game absent; no combination with masochistic-pseudo-sadistic feelings; the only feeling is that a necessary but disagreeable job had to be performed.	8. Element of infantile game present, combined with masochistic ?pseudo-sadistic excitement, usually repressed.
9. Success expected.	9. Defeat unconsciously expected.

THIRD LABOR: *Maintaining infantile megalomania.* The psychic masochist faces a seemingly insoluble conflict. On the one hand, his is an overdimensional megalomania; on the other hand, he aspires to an inner aim in direct opposition to his megalomania, since he unconsciously desires humiliation, defeat and ignominy. How does he manage? He finds a solution both ingenious and unique: on the condition that he himself creates his defeat, he accepts humiliation. In this way he maintains this inner fiction: "This idiot (the punisher) thinks he is kicking me. That's only what he thinks; what I know is that I, through my initial provocation, *made* him kick me!"

Here is an example in concrete terms:

Two business men are in conference. One of them rings for his secretary and asks her to bring a pertinent file. She "makes the mistake" of bringing the wrong file, and when she attempts to correct her error repeats the mistake again, and yet again. After the third attempt her boss loses his patience and gives her a humiliating dressing-down. The other man finds his own impatience vanishing under a sudden influx of sympathy for "the underdog." That sympathy is superfluous if the girl is a psychic masochist. If she is, she repeatedly creates provocations which turn her boss into an instrument for achieving her unconscious aim of humiliation. She demotes him to an inanimate punishment machine, and inwardly thinks herself in control.

FOURTH LABOR: *Pseudo-moral connotation.* I suggested the term, "pseudo-moral conno-

tation in neurotic symptoms," to designate a mechanism in which actual educational dicta communicated to the child are unconsciously twisted around in such a manner that a reduction ad absurdum results. The technique consists of taking these dicta literally and out of context, applying them at the wrong time and place and with the wrong intention. The upshot is that this inner fiction can be maintained: "You, parents, said so yourselves!"

This procedure is a defensive measure of the unconscious ego, and part of the unending tug of war which the neurotic fights with his inner conscience. Since the superego uses the ego ideal (which incorporates parental dicta and also the child's megalomaniacal and inflated expectations for his future) as a yardstick against which to measure the ego—and invariably finds it wanting, there is no way of preventing the ego from using the same material defensively. The unconscious ego turns the tables and proves that the neurotic did not transgress the enshrined dicta. A great deal of sophistry is applied in the process.

In the analysis of a good-looking girl, pathologically overweight, many discussions revolved about the psychological reasons for her inability to reduce. Her voracious eating was an inner defense against reproaches leveled by her superego. Accused of masochistically wishing to be refused, she retorted: "On the contrary, I wish to get. See how much I eat!"

Pointing out these facts helped little, simply because the patient retreated behind the blank denial that she overate. To test her statement, I suggested she call in a good friend (a slim woman) as character witness. This friend had originally pushed her into analysis; they frequently lunched together. The witness' reply was disconcerting to the patient; the friend said, "Tell him you eat like a pig." The patient protested. Since they were still at the lunch table, she could present a telling argument: they had identical checks. But the friend disposed of that argument at once. Their orders had been the same, but the patient had cleaned her plates while the friend had eaten only half of each dish. "But I had to eat everything," explained the patient, "my mother told me it is bad manners to leave anything on the plate."

The patient forgot to mention that this dictum dated from her eighth year, when she needed to be "fattened up." Thus an educational command pertaning to *fattening* was put to use nearly 25 years later, while she was *reducing*.

In the next three months of analysis the patient lost 35 pounds.

Another patient, a masochistically regressed woman, remembered the following incident after this mechanism was explained to her in analysis:

Her father, a rigid educator, had specifically forbidden his children to pick fruit from his prized apple orchard. At harvest time he discovered that the apples had been left on the trees but that this girl had climbed the trees and nibled at the apples as they hung from their stems. The father's severe reprimand was countered with an impeccable argument: "You didn't say anything about eating. All you said was—'Don't pick!' "

Still another patient, a young man with severe masochistic personality neurosis, suffered from inability to work and from a potency disturbance. He persisted for a long time in his refusal to take a certain technical examination. "Take this idiotic examination!" his infuriated father would demand. "I am not thoroughly prepared yet," the son would constantly reply. The superficial layers of his examination fear were easily explained. His father had tried to teach him the alphabet at the age of four. Dissatisfied with the child's progress, the father repeatedly complained that he was not "thorough" enough in his studies. Two decades later, the son executed his by then full-fledged masochistic neurosis (also manifested in his wish to fail the examination) via pseudo-aggression: "Well, I'll give you thoroughness—till you're blue in the face!" The fact that the student damaged himself more than he did his father is another story. He accepted the blame for the lesser crime of pseudo-aggression, denying psychic masochism, and inserted a pseudo-moral connotation into his secondary defense, asking, "What's wrong with doing as father said, and being thorough?" Time, place and occasion were of course different, so that he reduced his father's demand to absurdity—another characteristic quality of the pseudo-aggressive defense.

The mechanism here described is of prime importance in analysis of psychic masochists. The enigma of psychic masochism within the specific individual cannot be solved without finding the exact inner prop upon which that sector of neurosis rests.

FIFTH LABOR: *Carefully taking aim—the double requirement of a bull's eye and hitting below the belt.* If the psychic masochist is to make his provocations effective, he will have to be a sharpshooter who always hits his target, and also know how to choose the

right target: the weak spot in the person he wishes to provoke. An amazing amount of clever though unproductive thinking is required.

A snobbish patient, a man of professional standing, worked in a charitable organization where the atmosphere was haughty and patronizing. He explained his activity there as "a hobby," although he obviously enjoyed his contacts with the "high class people" connected with the organization. He was at that time chairman of some routine committee; the previous year he had been chairman of the program committee, a more important post. He had done an excellent job, securing speakers of prominence for the organization. The organization had a rotating policy for chairmanships, so that he moved on to another committee at the end of the year and someone else took over programming. At the annual membership meeting, where all committee chairmen reported to the full membership, my patient's successor as head of the program committee reported dull and mediocre accomplishments, but by putting on airs and embellishing facts attempted to show that he had done wonders. My patient was the next speaker. Although dead seriousness was the rule in this organization and witticisms were taboo, he began his report with this joke:

A counterfeiter just released from prison after serving a ten-year sentence formed a partnership with a gangster and returned to his old profession, producing bogus twenty-dollar bills. All went well until the partners discovered that, by some inexplicable error, the figure printed on the bills is not 20, but 18. "My God!" exclaimed the partner. "That's murder—who will taken an eighteen-dollar bill?" The counterfeiter tells him that all is not yet lost. "We can unload in some hick town—they won't know the difference." The partners arrive in a hick town, enter a bar and order drinks. They hand over a bogus eighteen-dollar bill in payment, and ask for change. "Sure," says the bartender. "Do you want two niners or three sixers?"

The joke reduces the forgers to absurdity by a deadpan pretense of acceptance: if there are eighteen-dollar bills, there are also nines and sixes. One absurdity eradicates the other.

By applying this story in the specific situation, the patient used a clever innuendo to tell his audience: "The preceding speaker considers you stupid hicks who cannot tell the difference between the bogus and the genuine. But you are too smart to fall for this trick."

I asked the patient when he had thought of the joke and whether he had prepared it in advance. "Oh, no," he said. "I thought of it during the few seconds it took me to reach the rostrum from my seat." I asked why he had told the joke; he told me that "it seemed such a good joke." He was "surprised" when I explained its meaning. Rather depressed, he then admitted that the speaker he had attacked turned his back on him during the dinner party that followed the meeting. At the time the patient had thought: "What does this fool have against me?" His campaign to "win friends and influence people" subsequently suffered a severe setback, since many of the officers of the organization became "rather cool" to him in the following weeks and months.

SIXTH LABOR: *Race against time lest tension develops.* Innter tension can be defined as a painful and uncomfortable feeling arising at moments before the inner alibi is presented to, and half-accepted by the inner conscience. I have recently devoted a volume to this topic (*Tensions,* Liveright, New York, 1960).

All the mental operations included in the "six labors" of the psychic masochist go on intrapsychically with a speed unimaginable to conscious thinking. Once more, respect is due this unconscious process.

Although the modern Hercules—the psychic masochist—has reduced by half the original "twelve labors," *inwardly* he performs a feat greater than that of the Greek hero. The ironic similarity and dissimilarity goes even farther. *Objectively,* Hercules was the innocent victim of a woman (Hera) who hated him even before he was born; the modern Hercules *subjectively* considers himself the victim of the Giantess of his individual nursery (on to whom he projects all possible sinister designs), although *objectively* most mothers love their children. Philosophers may marvel at that contradiction.

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Feasibility of "Browsing" Double Blind Studies

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The clinical evaluation of a new drug usually begins with a "browsing" study. "Browsing" is defined by Feldman[1] ". . . as that field of inquiry that lies between animal experimentation and pre-clinical testing, on the one hand, and the area of controlled clinical trials and the testing of specific hypotheses on the other." A clinician distributes a new drug to a variable number of patients, areas of usefulness are localized, dosage levels are varied, toxic effects are noted and clinical response is evaluated. Usually controls are not used in any form. It is called "browsing" because it is not controlled and consists of an initial evaluation under the grossest of scientific conditions. These studies are often used much beyond their period of usefulness, and after more definitive studies are indicated. The history of medical treatment and drug evaluation reveal that these studies frequently result in greater enthusiasm for the drug than is warranted by the facts.[2] The vast majority of these favorable reports are due to the placebo effect rather than to any intrinsic property of the new medication. Later, with waning enthusiasm or negative results, with and without controlled studies, the drug loses its place in the therapeutic armamentarium. The process is expensive, time-consuming and inefficient. More effective methods of evaluation are necessary, especially with the ever-increasing supply of new drugs.[3]

The double blind method is now being used and advocated as one possible answer to this problem. Although it was first used in embryonic form as long ago as 1906,[4] its use in psychiatry has become popular only since the introduction of the tranquilizing drugs in 1954. Some oppose the use of the double blind method, feeling that it is less reliable than a clinical appraisal, that it adds little information, that it can be dangerous, that it is generally unnecessary for the evaluation of drugs or that it is indicated only in certain specific situations.[5-10] Others are of the opinion that the placebo effect can be minimized or obviated by this procedure, or that it is the only method which can approach reliable discrimination between effective and ineffective drugs.[3,11-24] May of the pitfalls are enumerated.[3,8,23-26] One author in favor of this procedure under some circumstances has suggested that it is too complicated and raises more questions than it answers.[7] Others argue that it requires a great deal of time, effort, energy and personnel,[13] or that it requires well-trained personnel and too much time and expense for use in state hospitals.[1] Since the state hospital houses the sickest and the largest number of patients, it is very important that drug evaluation studies be done at these institutions. In addition, they are the only places in which large numbers of patients are available for study.

These considerations led to our interest in evaluating the feasibility of conducting a double blind procedure in a state hospital as one possible method of obtaining more reliable information about the usefulness of drugs.

A "browsing" double blind study of iproniazid in chronically depressed, state-hospitalized, geriatric patients was designed and executed.[26] The study was designated as "browsing" because its essential character was similar in many ways to the usual initial drug evaluation studies by clinicians. The time available to the investigators in conducting this study was limited; the study was incidental to regular clinical duties; there was no financial assistance; there were no specially trained personnel (except for statistical consultation) : and the study could not be planned and executed with consummate thoroughness. It differed from many

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"browsing" investigations in that a considerable amount of information about the chosen drug was already available and in the use of the double blind method and other controls.

Prior to the study, the seven individuals involved in evaluating or caring for the patients—three psychiatrists and four attendants—were asked to record whether they thought the double blind method was an effective method for the evaluation of drugs. Following completion of the study, they were again asked to record their impression about the effectiveness of the double blind method and whether extra time or difficulty was encountered in conducting the study.

Following completion of the study, all the psychiatrists felt that the double blind method was more valuable than a non-controlled or simple placebo-controlled study. One of the psychiatrists, although previously dubious about the merits of the double blind method, at the end of the study became convinced of its potential usefulness and advantages. A second psychiatrist became more enthusiastic about the procedure post-study compared to pre-study. The third psychiatrist, who was most in favor of the procedure pre-study, continued to be convinced of its virtues post-study. Two of the attendants thought the double blind method to be superior to other methods of evaluation. One attendant thought it unnecessary, and one did not understand the procedure.

The three psychiatrists were of the opinion that the time required for the double blind study was minimal. It did not interfere with other clinical responsibilities. It was thought that the double blind method required little if any extra time compared to a simple study. It was felt that the determination to execute a double blind study was more essential than a consideration of time, effort or other possible difficulties. Three of the attendants felt that the double blind method, in comparison with any study, did not add extra work to their schedules, take them away from patient care or interfere with other duties. In addition, the extra time required by the study, compared to no study, was minimal. Two attendants thought that the study required extra time at the beginning, especially because the medication had to be given t.i.d. Although this became routine and required less time after a while, one attendant felt that, in general, the study required more of her time than would a simple study without placebos, and if the medication had been given once per day instead of t.i.d.

Our impression is that the "browsing" double blind method is feasible in large state hospitals, and that the determination to execute a double blind study is more essential than other considerations.

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The Clinical Effect of Nor-Eth-Androlone (Nilevar) on Incontinence of Mental Patients

(Preliminary Report)

GEORGE VLAVIANOS, M.D., and LUDWIG FINK, M.D.

Nor-eth-androlone (17-a-ethyl-17-hydroxy-19-nor-4-androstan 3-one) Nilevar, a new synthetic compound belonging to the endrogenic steroid group, has a useful counter-balance of anabolic to androgenic properties. This compound, described at a conference on its clinical use,[1] as exerting minimal androgenic activity, also acts as a "protein anabolic" hormone, promoting the synthesis of muscle proteins from the nitrogenous food and having in addition a "myotrophic effect." The latter was experimentally determined in animals by the levator ani method.[2] Questions have been raised whether the myotrophic effect would apply to the levator and perineal muscles in humans. Would there be a lessening of incontinence of feces and urine clinically evident? As the authors see a large group of deteriorated mental ward patients incontinent as to urine and feces, a clinical trial with this medication was made.

Procedure

A few doses were tried in September 1957, on an extremely regressed and incontinent schizophrenic patient (No. 1). He would sit idle during the day, urinating in his clothes five or six times and soiling with feces. He would also wet and soil during sleep. Given 10 mgs. of Nilevar T.I.D. for seven days he stopped wetting and soiling during the day, began to help with ward chores and showed improvement in his general behavior while on the drug.

Encouraged by this result, 12 patients, including No. 1, were placed on a trial supply of the drug. Eleven of this group consisted of the most severely affected, chronically incontinent from 1 to 55 years, with various mental diagnoses. Case No. 12 was an adult hebephrenic praecox, with incontinence of only one month, although hospitalized for eight years.

Table I lists the relevant clinical data of these 12 patients. They were weighed before and after the course of Nilevar in 10 mg. doses Q.I.D. Medication lasted from 17 to 53 days (in this group) with an average of 25 days. All 12 cases had been screened for interfering bladder or prostata lesions. There were no alterations in their pre-existing fluid and dietary regime or in the nursing care.

Laboratory Examinations: Blood urea nitrogen, non-protein nitrogen, total cholesterol, total protein, albumin and and globulin ratio, serum alkaline phosphatase and serum

From Kings Park State Hospital, Kings Park, New York.

Nilevar is the trademark of Searle and Company.

TABLE I

Clinical Data of Incontinence, Duration of Treatment and Results of Incontinence

Case No.	Age	Mental Diagnosis	Duration of Incontinence	Treatment	Incontinence
			yrs.	days	
1	44	Dementia Praecox Paranoid Type	5	44	Improved
2	63	General Paresis Tabetic Type	15	25	No change
3	60	Manic Depressive Manic	18	25	No change
4	64	Psychosis *c* Meningo Encephalitis (Syph.)	21	53	Improved
5	46	Dementia Praecox Paranoid Type	2	17	No change
6	56	Dementia Praecox Simple Type	6	17	No change
7	60	Psychosis with Mental Deficiency	55	17	No change
8	69	Dementia Praecox Paranoid and Alcohol Psychosis	4	28	Improved
9	76	Manic Depressive Manic	22	28	Improved
10	64	Manic Depressive Manic	1	21	Improved
11	45	Psychosis due to Unknown or Heredity Cause (Hemiplegia)	3	21	Improved
12	35	Dementia Praecox Hebephrenic Type	1 mo.	20	Improved

bilirubin all showed no change during treatment.

All episodes of wetting and fecal soiling were registered on a 24-hour basis.

Results

Seven of the group had a fair to good response with no incontinence from three to seven days after the start of medication. They remained continent during the treatment. Three of them continued to be continent after medication was stopped, two for the following five days and one for 21 days. Five patients showed little or no influence on their pattern of incontinence. There were no side effects nor any change in weight. The original patient (No. 1) showed improvement in his schizophrenic behavior as his incontinence lessened.

Evaluation

The number of patients was small and limited to male adults only, the time of trial too short to evaluate conclusively the temporal usefulness, long term action and potential side effect of this drug on bladder and bowel incontinence. No experience could be gained as to the efficacy of the drug on female incontinent patients and incontinent children. The improved behavior of patient (No. 1) diagnosed as schizophrenia, under the drug is noteworthy and further investigations are warranted. The serious impact of the "incontinent patient" on the hospital as well as the community from social, hygienic, and medical points of view makes us consider this condition a major medical and public problem. The limited attention paid to this topic in the literature convinced us that this preliminary report would be of interest.

Summary

The effect of nor-eth-androlone (Nilevar), a synthetic "protein anabolic, myotrophic" steroid, on the incontinence of 11 chronic psychotic males and one acutely incontinent adult hebephrenic praecox, was studied for a period of 17 to 53 days. Seven of them had a favorable response. Five patients showed scattered or no influence on their pattern of incontinence. The serious social impact of the "incontinent patient" is stressed.

Acknowledgments: The authors gratefully acknowledge the cooperation of Charles Buckman, M.D., Director of Kings Park State Hospital; of J. Rothery Haight, M.D., Assistant Director; of Reuben Cares, M.D., Director of Clinical Laboratories; and of Mr. William Moore, Attendant in charge of Ward 55, Kings Park State Hospital.

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Treatment of Alcoholic Patients with Triiodothyronine

JAMES H. SATTERFIELD, M.D., and SAMUEL B. GUZE, M.D.

In 1956 Curt P. Richter[1] reported that rats given a free choice of drinking a 10% solution of alcohol or water would consume about 15 ml. of the alcohol solution per day. When thyroid powder in 1% concentration was added to the food the rats almost immediately stopped drinking the alcohol solution, drank more water and ate more food. Concentrations of thyroid powder as low as 0.04% were effective in reducing the alcohol intake. Thyroid powder had the same effect on the appetite for wine, beer and whiskey. Thyroxine and triiodothyronine were equally effective. This suggested the possibility of reducing the alcoholic intake in man by treating the alcoholic patient with thyroid preparations.

The purpose of this study was to determine if there were any gross differences in clinical response of alcoholic patients to triiodothyronine and a placebo.

Method

Our study comprised 24 patients; 21 male patients, who volunteered for a six-week period of drug therapy were selected from the alcoholic in-patient service at Malcolm Bliss Hospital. The remaining three cases were private patients who offered to participate in the six-week trial period of treatment. All of our cases were inveterate drinkers of long standing. Half of the patients received two 25 microgram tablets of triiodothyronine per day and half received two identical placebo tablets per day. Neither patient nor the investigator knew which patients were receiving the active drug. All patients were given a one or two week supply of medicine with instructions to take one tablet twice a day. Patients were seen for follow-up at one or two week intervals.

Results

Among the twelve patients who received triiodothyronine there were two who returned for six weeks. One of these was not drinking at the end of that time and the other took the drug regularly but continued to drink and was drunk when last seen. One patient returned at the end of two weeks and was not drinking. Two patients returned at one week, one was not drinking and one was drinking. The other seven patients did not return at all. Of the twelve patients receiving the placebo, one attended the full six weeks, one for three weeks, one two weeks, and one only one week. None of these patients had resumed drinking. The remaining eight placebo patients did not return at all.

Conclusions

In this pilot study there was no difference in the clinical response to triiodothyronine and to placebo therapy. It should be pointed out that the majority of the alcoholic patients were severe alcoholics with a long history (average of 18 years) of drinking. Perhaps a study with a group of less severe alcoholic patients is indicated.

From the Department of Psychiatry and Neurology, Washington University School of Medicine and Research Laboratories of Malcolm Bliss Mental Health Center, Saint Louis, Missouri.

The drug triiodothyronine was supplied by Warner-Chilcott Laboratories, Morris Plains, N. J.

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Book Reviews

CEREBROSPINAL FLUID: DYNAMICS IN HEALTH AND DISEASE. David Bowsher, M.A., M.D. Charles C. Thomas, Springfield, Illinois, 1960. 67 pp. $4.75.

This concise and well-written monograph offers a review of old theories and presents new theories and hypotheses about the formation, flow and absorption of cerebrospinal fluid. The author proposes the concept of cerebrospinal fluid as a single entity. He bases this conclusion on the results obtained by Sweet, *et al.*, who used isotopes of naturally occurring cerebrospinal constituents for measuring production and absorption of cerebrospinal fluid. The author's own histioautoradiographical studies are compatible with this theory.

With the help of ingeniously constructed simple models, the author continues to build up a theory about the mechanism of absorption, supporting it with isotope studies (NA 24). Theories concerning the development of hydrocephalus are offered, with defective leptomeningovascular absorption seen as the main factor in the development of hydrocephalus.

The author concludes with reflections on the breakdown of the blood-brain barrier, either locally or generally. He feels that edema, which can bring about a decrease in cell packing density and by so doing allow free exchange to occur between plasma and cerebrospinal fluid, is the causative factor in the barrier breakdown. The monograph includes an impressive bibliography of 179 references.

The reviewer is in agreement with Dr. Bowsher in his statements that much of the theoretical presentation is still based on slender evidence. He cautions that different techniques may lead to different findings and interpretations, and results obtained with one technique are prone to varied explanations. This is nonetheless a valuable contribution and well worth reading.

Cornelius Boelhouwer, M.D.

NEUROPHARMACOLOGY — Transactions of the Fourth and Fifth Conferences (September 1957, May 1959); Sponsored by the Josiah Macy, Jr. Foundation; Harold A. Abramson, M.D., Editor; two volumes (285 pages and 251 pages); $5.00 each volume.

During the past 15 years the Josiah Macy Jr. Foundation has sponsored a series of conferences, the participants of which are recognized authorities or specialists in the fields of medicine and health. The goal of the conference program is the promotion of communication, the exchange of ideas and the stimulation of creativity among the participants. The purpose of publication of the Transactions of the meetings is to share, as far as possible, the conference process with a larger audience than could participate personally in the discussion.

These two volumes report the recorded sessions of discussion held by a group of investigators in the field of basic neurophysiology and biochemistry. Most of the discussion is too highly technical for the clinician, but is valuable to give the clinician an understanding of and an appreciation for the techniques and problems of research methodology.

In the 1957 conference four papers were presented: "The Effect of Respiratory Poisons and Anoxia on Siamese Fighting Fish in Relation to LSD-25 Reaction" by Harold A. Abramson; "Clinical Studies with Taraxein" by Robert G. Heath; " 'Stop' and 'Start' Systems" by John C. Lilly; and "Some Relations Between Chemical Structure and Physiologic Action of Mescaline and Related Compounds" by Gordon A. Alles. The paper on Taraxein was of particular interest since it unfolded the drama of Dr. Heath's defense of his findings and methodology on experiments with Taraxein, a substance which he fractionated from the sera of schizophrenic patients and which he claims can produce schizophrenic symptoms when administered to nonpsychotic volunteers. It is heartening to see the findings, feelings, criticisms, doubts and agreements of the participants in this discussion so openly published.

The second volume which reports the Transactions of the 1959 Conference is editorially better than the first in that it presents summaries, abstracts and bibliographies for the highly technical biochemical subjects. There are four topics present in this volume: "Amine Metabolism and Its Pharmacological Implications" by Sidney Udenfriend; "Central Action of Chlorpromazine and Reserpine" by Keith F. and Eva K. Killam; "Physiological Fractionation of the Effect of Serotonin on Evoked Potentials" by Werner P. Koella; and "Biochemical Sites of Action of Psychotropic Drugs" by Bernard B. Brodie. The psychiatric clinician can benefit greatly by reading these discussions and gaining a broader knowledge of the fathomed and unfathomed biochemistry of such physiologically important substances as serotonin and norepinephrine which are so intimately related to the possible actions of some of the tranquilizer and antidepressant drugs so widely used in psychiatric practice today.

William W. Zeller, M.D.

NEUROLOGICAL AND NEUROSURGICAL NURSING, Third Edition, by de Gutierrez-Mahoney and Carini. C. V. Mosby Company, 1960, St. Louis.

This is the third edition of the well-known *Neurological and Neurosurgical Nursing* which has been a standard text in schools of nursing since the first

edition appeared in 1949. The format of the book has been modified, the bibliography updated, treatment and nursing care emphasized.

This edition is new in that the material is directed even more towards nursing care, but the reader will find that the contents remain much the same as in the second edition.

Psychiatric aspects of the neurological patient has been superficially treated and the reader is urged to consult other sources for help in this area.

The excellent illustrations of the previous edition have been retained and contribute much to the teaching value of the text.

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